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(Continuing the Journal of the Allied Dental Societies)

A journal of stomatology; devoted to the advancement and dissemination of knowledge pertaining to the teeth and mouth, and to their relations to the body as a whole

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A Summary of the Titles of the Papers, and the Names, Degrees and Professional Relationships of the Authors, With Abstracts or Interpretations of the Original Matter In Each Communication

These abstracts are neither editorials nor criticisms; they do not contain comment beyond the scope of the papers to which they refer. They are intended solely to give the reader a general though clear idea of what the corresponding authors have chiefly to say.

[*These abstracts are reprinted in this issue, with the title page and other introductory matter intended for bound copies of the volume (IV) for 1922. In the binding of the volume, this insert should be rejected.*]

Papers in the Main Body of this Number

A discussion of problems concerning pulpless teeth and pulp devitalization. Authentic information and representative opinions, compiled from responses to a questionnaire. (*Fourth and concluding part.*) By Elmer S. Best, D.D.S., Minneapolis, Minnesota..... 379

In this concluding part of his study, Best presents, under a copy of each of the fifty-two questions in his questionnaire, a summary of his own general conclusions from the facts and opinions respectively stated by the correspondents, and published successively in the first three parts, in this volume. This fourth part is, in effect, an abstract of the preceding parts, and accordingly presents the gist of the entire compilation.—(W. J. G.)

Notes on the minute osteology of the jaws. By Josef Novitzky, D.D.S., San Francisco, California..... 399

Novitzky reports the results of "a study of the network of osseous trabeculae composing the spongy tissue of the mandibular and maxillary bones of man." He differentiates between calcification and ossification; emphasizes certain roentgenographic distinctions; alludes to the causes and significance of eburnation, and to the import of fibrous marrow; notes that regions of localized eburnation are common; finds that calcification resulting from an inflammatory reaction to a low-grade infection is a pathological process; and discusses granulomata and current methods of filling root canals.

Novitzky sums up, under general conclusions, as follows: "Every careful study in the field of the osteology of the jaws reinforces the fact that every dead tooth is infected; that no method of 'treatment' has succeeded in rendering a dead tooth permanently sterile and harmless; and that the only safe procedure, in the case of a dead tooth, is surgical removal of the tooth and surrounding regions of infection."—(W. J. G.)

Studies on the relations of types of diet to dental caries. A statistical study of children under school age, relating to two dietetic theories on the etiology of dental caries. By RUSSEL F. RYPINS, M.D., Department of Metabolism, Deaner Dental Institute, Kansas City, Missouri..... 405

In this study, on children under six years of age, Rypins has given special attention to two dietetic theories of the etiology of dental caries. One is the theory that "indulgence in sugars leads to dental decay;" the other is the "'physiological' oral hygiene thesis as defined by Wallace," to the effect that "caries is initiated by acid fermentation of sticky foods which adhere to the teeth. Cleaning the teeth after meals prevents such fermentation. The same result is effected by mastication of 'natural' food-stuffs which are detergent, and preferably acid in reaction, at the termination of each meal. 'Physiological' oral hygiene so maintained will prevent dental caries." *Neither of these theories is supported by Rypins's findings.*

The study "included examinations of 1,068 children of preschool age" for "gross caries, obvious to careful direct inspection without dental instrumentation." The dietary conditions of the children under examination were ascertained from the mothers, and recorded in "an arbitrary food classification." The paper is a statistical report. "It is definitely understood that proof or disproof, of the absolute validity of hypotheses which relate to dental caries, cannot be established by statistical methods alone." Numerous details are presented, and the data are effectively tabulated and graphed. The many sectional headings help the reader to follow the author through the numerous phases of his discussions. The gist of the author's findings is given in the following quotation:

"Since the theories studied are not borne out at all by the clinical-statistical method employed, their validity must be held in some doubt until otherwise fortified with equally reliable

methods or data supporting them. This is not a contention that the theories have been disproved or completely invalidated. It indicates only that their validity has been challenged. Until we have results of further careful research upon them, we must be cautious not to be converted by the mere pictorial impressiveness of the theses. The everyday findings of wholesale decay of teeth in a people living on our artificial dietaries, or who lavishly indulge in candies, jams and sugars, should not alone, or without carefully controlled comparison series, persuade us prematurely to conclusions on this important question."—(W. J. G.)

Blood chemistry as a diagnostic aid in focal infection. By RICHARD N. DENIORD, M.D., Buffalo, New York..... 435

The author states that the proportion of uric acid in the blood is a diagnostic index of the existence of focal infection, after exclusion of all other factors that increase the content of uric acid in the blood. The "other factors productive of a high uric-acid value for the blood, aside from nuclear degeneration, are comparatively easy to determine, namely, leukemia, primary anemias, cachexias from whatever cause, and massive doses of x-ray or radium."

According to De Niord, "elimination of all foci of infection is followed invariably by a return to normal of the uric-acid value for the blood. Failure to eliminate all foci prevents the return to a normal uric-acid value for the blood." The author states that the chemical findings do not "determine whether the focus is present in teeth, tonsils or sinuses," but they do "indicate the presence or absence of *foci in the body*." The chemical findings for uric acid in the blood "are also extremely valuable," the author believes, "as a check on the thoroughness of eradication of the foci found, for, as long as any focus is present, the uric-acid findings will not return to the normal level."

The author discusses the focal-infection theory in general; the origin of uric acid in the blood; and the various influences that increase the proportion of uric acid in the blood, independently of focal infection. He presents case histories, uric-acid graphs, and summaries of collateral analytic data. (See the discussion of the related paper by McCall, in the proceedings section, p. lxxxi.)—(W. J. G.)

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SCIENTIFIC PROCEEDINGS OF DENTAL AND STOMATOLOGICAL SOCIETIES

Oral diagnosis. By JOHN OPPIE MCCALL, D.D.S., Buffalo, New York lxxi

The author discusses broadly the "theory that systemic disease may be caused by oral foci of infection." He is "firmly convinced of the fundamental soundness of the focal-infection theory," discusses the theory and its application in practice, and alludes to the practical utility of De Niord's diagnostic index (see the preceding abstract). In some remarks on radiographic evidence he writes: "My present contribution to the interpretation of radiograms lies chiefly in calling attention to the importance of periapical lesions other than the classical circumscribed radiolucent region." He also invites "attention to those parietal lesions which may call for tooth extraction, in cases of metastatic disease," and refers to such matters as impacted teeth, unextracted root tips, and "residual infection," in their relations to focal infection.

McCall reviews his personal experience with focal infection and presents a statistical review of the results of extraction of teeth for patients in his own practice. His own experience and these "results" fortify his faith in the soundness of the focal-infection theory. He believes "that it is both theoretically and practically possible to remove the dental pulp and fill the canal with safety to the patient;" but he also believes "that the best method of accomplishing this has yet to be devised." (See the succeeding abstract for references to a discussion of this paper.)—(W. J. G.)

Discussion of the paper by John Oppie McCall, on "oral diagnosis." By E. E. SMITH, PH.D., M.D., and M. L. RHEIN, M.D., D.D.S., of New York City; and Dr. MCCALL lxxxi

Smith discusses chiefly the "symptoms that may be attributed to oral focal infections," and says that, "from the foregoing it is clear how uncertain is any positive conclusion of an etiological relation of periapical foci to a symptom manifestation in an individual case." He doubts the diagnostic reliability of De Niord's uric-acid index (see page 435), and gives his reasons for a skeptical attitude in this relation.

Rhein agrees with McCall in the main, but protests against McCall's restricted use of the phrase "oral diagnosis," in a restricted sense, as the title for his paper.

In closing the discussion, *McCall* alludes in a general way to the dissenting opinions of Smith and Rhein, and agrees with Rhein on the full import of the phrase "oral diagnosis."—(W. J. G.)

Discussion of three papers (by Hinman and Johnston, on pp. 103–128 of this volume) on problems pertaining to pulpless teeth. By ARTHUR B. CRANE, D.D.S., of Washington, D. C.; RODRIGUEZ OTTOLENGUI, D.D.S., LL.D., and M. L. RHEIN, M.D., D.D.S., of New York City; and HARRY B. JOHNSTON, D.D.S., of Atlanta, Ga..... lxxxix

Crane contends that a pulpless tooth is not necessarily a dead tooth, endorses efforts to save pulpless teeth under proper conditions, alludes to the current misuse of the word "ionize," mentions electrochemical phenomena involved in electrolytic medication, discusses methods of filling root canals, and offers constructive criticism in general.

Ottolengui feels that "pulpless teeth, even when diseased, can be saved;" believes the "idea of sterilizing the apical region" has been overemphasized; and considers that it is "impossible to sterilize any enclosed cavity in the body." He cites and illustrates the cure of a "hopeless" case by electrolytic medication.

Rhein's remarks include much that recently appeared in his paper on a related subject in this volume (page 327). He is deeply gratified by the fact that all concerned in this discussion favor efforts to save pulpless teeth. He alludes to the difficulties inherent in the execution of a proper root-canal technique; and comments adversely on the suggested variations from his own procedure, which he believes is "better than any other that has ever been shown" to him.

Johnston responds to the criticisms in accord with the remarks in his papers (pp. 109–128). To the discussion is appended a series of 30 radiograms, with corresponding legends, illustrative of the papers by Hinman and Johnston under discussion.—(W. J. G.)

American Academy of Periodontology. Proceedings of the Ninth Annual Meeting: Secretary's general report, President's address, and reports of Committees. By ARTHUR H. MERRITT, D.D.S., Editor..... cv

I. The *secretary's report*, by J. Herbert Hood, presents a general outline of the proceedings of the meetings in Cincinnati, Ohio, on December 4–6, 1922.

II. The *president's address*, by Clyde M. Gearhart, is chiefly historical in character—on the development of the Academy. He suggested that "at least a part of the activity of this organization should be directed toward investigation of the standards prescribed for oral hygienists, to the end that they may become of even greater service to the public."

III. The *report of the Committee on Fellowship*, presented by Paul R. Stillman and Clyde M. Gearhart, recommended, for election to Fellowship, Drs. Harold Keith Box, Grace Rogers Spalding, Gillette Hayden, and John Oppie McCall.

IV. The *report of the Committee on Research*, presented by Andrew J. McDonagh, considers the relation of diet to dental conditions. The responses to a questionnaire on this subject are summarized, and the conclusions drawn "that certain food habits, which some enthusiasts claim are the cause of dental and periodontal troubles, cannot be relied on to prove these theories. . . . . One outstanding fact seemed to be evident throughout (the responses to the questionnaire), namely, the *almost universal connection of worry with dental periclasia.*"

The paper by De Niord (page 435) is a part of the proceedings of the Academy at this meeting.—(W. J. G.)

Dental education. By ALFRED OWRE, D.M.D., Dean of the School of Dentistry, of the University of Minnesota, Minneapolis, Minnesota..... cxvii

An historical paper. Owre traces the lines of progress from ancient times, through the middle ages and the period of the revival of learning, to the "rise of modern dentistry." He compares the developments in medical education with those in dental education, alludes to "recent progress in dental education," and discusses "dentistry as a branch of medicine."—(W. J. G.)

A Statement to Dentists

Regarding a Very Valuable New Book in Dental Science

Dr. William K. Gregory's book on the ORIGIN AND EVOLUTION OF THE HUMAN DENTITION, referred to on the next page, is the most complete, and the most effectively and profusely illustrated, work on the subject. It was written by the man who is most competent to do so. It will not be out of date in a year or two, for it deals broadly with fundamentals. Every dentist should study it.

Dr. Gregory's excellent book should be placed in every dental library. The students in every effective course in dental anatomy, dental physiology, and orthodontia, ought to learn the essentials of what the book presents. I recommend it for use as a *text book or reference book in every dental school.*

Dr. Gregory has given to the JOURNAL OF DENTAL RESEARCH *all his property rights in the book. The proceeds of its sale will be donated by him to the permanent Endowment Fund of the* JOURNAL OF DENTAL RESEARCH.

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Dr. Gregory's book is exceptional in its scholarly, scientific, and practical aspects. It is not only a splendid contribution to dental science but a notable tribute to dentistry. I commend the book, and Dr. Gregory's rare generosity, to the consideration of every dentist.

See the reviews of the book, on pages (39) to (47), inclusive, of the general matter at the back of this issue.

WILLIAM J. GIES

THE ORIGIN AND EVOLUTION OF THE HUMAN DENTITION

A Palæontological Review

BY

WILLIAM K. GREGORY, A.M., Ph.D. (Columbia)

Curator of the Department of Comparative Anatomy, American Museum of Natural History, New York; Associate Professor of Vertebrate Palæontology, Columbia University

Illustrated with 15 plates and 353 figures

6 x 9 Cloth, gold stamped 548 pages

Detailed Index Full Bibliography

The Origin and Evolution of the Human Dentition is a masterly review of the palæontological facts relating to the origin and evolution of the teeth in man, by one of the best informed investigators in this field. The book presents a complete summary of the main facts in the palæontological record on the origin and evolution of human dentition; and will be universally regarded as the most notable and successful effort to epitomize this information, to reveal its full evolutional import, and to suggest its practical significance. This review is a great fund of important facts and opinions in dental science, and must be STUDIED, particularly in close comparison of the text with the many exceptional illustrations, to be fully appreciated.

The author discusses broadly various important phases of human evolution in general.

Of special interest to dentists, physicians, palæontologists, odontologists, morphologists, and students of evolution and inheritance.

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Part I. Traces the stages of ascent from the Silurian fishes to the mammals of the Age of Reptiles.

Part II. Covers the stages from the Paleocene placental mammals to the lower primates.

Part III. Describes Nature's earlier experiments in evolving large-eyed and short-jawed primates.

Part IV. Discusses the dentition of the higher primates and their relationships with man.

Part V. Treats the later phases in the evolution of the human denture; gives a conspectus of the species and the chief races of mankind, with special reference to dental characters; and concludes with a general summary of twenty-six structural stages in the ascent of man and in the evolution of the human dentition.

A detailed index and a full bibliography are included.

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A DISCUSSION OF PROBLEMS CONCERNING PULPLESS TEETH AND PULP DEVITALIZATION

Authentic Information and Representative Opinions, Compiled From Responses to a Questionnaire[1]

(*Fourth Part*)[2]

ELMER S. BEST

Minneapolis, Minnesota

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[1] Best: *Journal of Dental Research*, 1919, i, p. 523; 1920, ii, p. 285; 1922, iv, pp. 1, 129, and 225.

[2] The *first part* was published in the *Journal of Dental Research*, 1922, iv, p. 1 (March); the *second part*, p. 129 (June); the *third part*, p. 225 (September). The four parts of which this compilation consists will be issued in book form on or about June 1, 1923.

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I. INTRODUCTION

The author presents, in the succeeding section, under a copy of each question in the questionnaire, a general summary of conclusions from the facts and opinions respectively stated by the correspondents, and published successively in the first three parts of this compilation.

The questions in the questionnaire are repeated in detail for the convenience of the reader, not only in the table of contents herewith but also in the text of this concluding part of the study.

II. COPIES OF THE QUESTIONS IN THE QUESTIONNAIRE; WITH THE AUTHOR'S SUMMARY, UNDER EACH, OF HIS CONCLUSIONS FROM THE FACTS AND OPINIONS RESPECTIVELY STATED BY THE CORRESPONDENTS

I. *In the treatment of vital deciduous teeth, where the decay is so extensive that its entire removal would mean the exposure of the pulp, what do you advise and what is your procedure? Also, how do you proceed where the pulp is already exposed?*

The consensus of opinion seems to be that every effort should be made to save the vitality of the pulp. Furthermore, there seems to be unanimity of feeling against exposure of the pulp in removing the involved dentin. Various methods are suggested for sterilization of the layer of dentin that has been left overlying the pulp. The inference seems to be that, if the pulp can be kept free from exposure, the chance of saving it is greatly enhanced.

II. *In the treatment of deciduous teeth where the pulps have become non-vital and infected, what is your procedure and what do you advise?*

While a certain percentage seem to favor extraction as the sole treatment for this condition, a majority make an effort, apparently, to differentiate between teeth to be saved by opening into, irrigating, and sealing root canals, and teeth that should be extracted. There is a noticeable absence of opinion that *all* such teeth should be saved. The use of the silver-reduction method appears to be the favorite where they are saved. There seems to be a common opinion that, if near the time for their exfoliation, the teeth involved should be removed.

III. *In case the root ends of permanent teeth are not completely formed and the pulp has decomposed, what is your procedure and what do you advise?*

Opinions seem to be about equally divided as to whether such teeth should be extracted at the beginning or an attempt made to save them. A small number advocate opening through the buccal or labial alveolar process and sealing the canal at the apex. There seems to be a general inclination to regard the saving of such teeth as questionable procedure; and there is a marked tendency to "keep an eye on them," if they are left in the jaw.

IV. *In case the root ends of permanent teeth are not completely formed, and the pulp is still vital but exposed, as a result of decay or trauma, what is your procedure and what do you advise?*

A great majority favor efforts to save the pulp, even (as in the case of no. 39) where the pulp has become infected. A few favor removal of the pulp or extraction. Attention should be drawn to reference, by some, to the desirability of leaving the apical pulp tissue undisturbed, in the event of removal of the pulp.

V. *In case the vital pulp is covered with a thin layer of decalcified dentine, what is your procedure and what do you advise?*

Opinions are almost unanimous that every effort should be made to save the vitality of the pulp. Opinions differ greatly as to whether a layer of decalcified dentin should be left overlying the pulp or whether the pulp should be exposed. A majority seem to favor leaving a little dentin in the bottom of the cavity as a covering for the pulp. Use of the silver-reduction method is freely mentioned.

VI. *At what age do you consider it good practice to begin bridgework that necessitates cutting into vital teeth? Do you consider that the location in the mouth is a governing factor?*

The age is set all the way from sixteen years to middle life. However, there seems to be a general opinion that complete development of the roots should precede the use of such work. Special attention is drawn to the number who advise some form of restoration that does not necessitate cutting into sound enamel. The *location* of bridgework in the mouth is seen from two points of view; some stating that it is a governing factor, others that it is not.

VII. *When permanent teeth of young patients are extracted on account of apical infection, do you retain the spaces with orthodontic appliances, use bridgework, or pay no attention to the spaces created by the extractions?*

A majority favor retaining the spaces. Special mention is made of removable appliances.

VIII. *Where deciduous teeth are extracted, do you, under any circumstances, advise retention of the spaces with orthodontic appliances, for the eruption of the permanent teeth?*

Opinions agree, in the main, that the spaces so created should be maintained, more especially if some time will elapse before eruption of the permanent teeth. A few are opposed to such a procedure.

IX. *What were the conditions that caused the death or removal of so so many pulps in the past? To what extent are these conditions now under control and pulp removal unnecessary?*

The responsibility is divided among physicians, dentists, and patients. Physicians have failed to appreciate the significance of dental caries and have neglected to advise patients to have carious teeth attended to. Patients fail to seek relief early enough, chiefly through fear of pain, which causes them to delay until carious cavities develop to the point where the pulp is involved. Also, many patients tolerate conditions of uncleanliness in their mouths that are conducive to development of caries. Dentists have been unaware of the results that frequently follow removal of the pulps of teeth, have used types of restorations necessitating pulp removal, and have failed to master the technic of preserving the vitality of pulps in teeth with large cavities. Relief can be assured (a) by educating the public, both adults and children, in the importance of clean, healthy mouths, free from dental caries; (b) when dentists perform dental operations without causing so much pain, use the newer types of dental restorations that do not require removal of pulps of teeth, and learn how to maintain pulp vitality in teeth with deep cavities; and (c) through the development of the specialty of pediadontia. The importance of deep study of each mouth, not only of individual teeth, is also mentioned. Great stress is laid on the practice of preventive dentistry as an important factor in keeping caries from progressing to a degree that requires removal of the pulp.

X. *What are the causes of pulp death over which we still have no control?*

Opinions are almost unanimous that trauma from accidental injury will continue to be one of the chief causes of pulp removal. Other causes over which we, as dentists, are not supposed to exercise control, are, neglect on the part of the patient, formation of deposits in the pulp chamber and canals, certain bad dental operations, and systemic conditions, including metastatic infection.

XI. *In a case where decay is in actual contact with a vital pulp, do you advocate (a) removal of all decay and attempted pulp capping; or (b) removal of the decay followed by pulp removal, (c) leaving just enough decalcified dentine to prevent actual exposure and giving the case a treatment? (c) What does such treatment consist of? How long have you practised it? Do you consider it a good procedure to follow?*

This question was asked because the author feels that much root-canal work has been due to the habit, of patients, of allowing large cavities to develop before seeking dental service, chiefly through fear of pain. The pulps in such cases have been removed.

While a number favor removal of the pulps in such cases, yet there is a noticeable tendency to save the pulps. The method of removing all dentin, partially or completely involved, seems to be giving way, in the opinions of many, to the plan of leaving even a thin layer of dentin over the pulp, on which is placed some material that will sterilize it. The results of this method of handling such cases, in the minds of many who use it, have been so gratifying that the attention of the reader is specially directed to it.

XII. *How early do you begin oral prophylaxis for your patients?*

The importance of early prophylaxis is shown by the unanimity of opinion to this effect. The prevailing idea is to start the supervision of the mouth as soon as possible.

XIII. *Outline your plans for preventing decay and pulp death in the teeth of young patients.*

The tendency to decay in children's teeth is recognized by all. The various valuable suggestions for the solution of the problem may be combined in one plan, as follows: Educate children and parents regarding mouth conditions in general. Teach a method for the adequate brushing of the teeth and use of dental floss; the latter, after each meal. Use an alkaline mouth wash. Areas showing decalcification of the enamel should be treated with silver nitrate. Occlusal

fissures should be thoroughly cleaned with pics and broaches, and silver nitrate solution flowed into them; others should be cut into and filled; some should have cement forced into them. There should be regular prophylaxis at the office of the dentist. The value of the work of the dental hygienist should be emphasized.

XIV. *Is it your habit to give the mouths of your patients prophylactic supervision during the time they are in your care? If so, how frequently do you see the patients for this work?*

Opinions agree that there should be regular supervision of all patients. The time between recommended visits varies from two weeks to a year, according to conditions in the individual mouth. It is important that patients should be able to keep all surfaces of all teeth polished, and free from mucous plaques and food debris; and also to keep the gums free from signs of irritation.

XV. *In deciding why the radiograms of some non-vital teeth show areas indicative of apical disease, and some do not, and also why some persons with such teeth display symptoms suggestive of focal infection while others do not, is it probable that different factors contribute to the two conditions at the ends of the roots? (Be sure to mention whether or not you believe the development of apical infection is solely a matter of diminished resistance on the part of the patient.)*

Many believe that diminished resistance on the part of the patient is a factor in such cases. Various other factors are mentioned, however, as contributing, if not responsible, agents for the condition.

XVI. *In the case of a non-vital tooth showing apical infection, outline methods by which such infection can be removed and the tooth retained. Do you consider that your procedure gives permanently satisfactory results?*

Opinions on this subject differ, although it is evident that all the correspondents aim to be as practical as possible. A majority attempt destruction of the apical infection through the root canal, and use various drugs for that purpose. The difficulty of doing this work on molars is mentioned by some. Quite a number favor a surgical method of getting rid of the infection—some by extraction and curettement, some by resection of the root tip. The uncertainty of the latter operation is mentioned by a number. One (no. 11) states that 90 per cent of such teeth have dead cementum, which can never again be united to vital tissue.

XVII. *Approximately what number of non-vital non-infected teeth would you advise leaving in any one mouth (a) in the absence of symptoms of focal infection; (b) in the presence of symptoms of focal infection? Do you make any distinction, in this regard, as far as age of the patient is concerned?*

The question is not sufficiently self-explanatory. It should convey the idea that, from a radiographic examination, we find no evidence of apical infection. The answers bring out quite clearly the fact that to extract pulpless teeth that show no apical disease, in patients who are in good health, is contrary to the practice of most of the correspondents. Where a patient is sick, there seems to be a marked tendency to have a consultation with the physician in charge before such teeth are removed. While there is uncertainty of opinion regarding the actual influence that pulpless teeth (non-infected so far as radiographic evidence is concerned) may have on a patient's health, there seems to be a strong feeling that an undesirable effect is not apt to occur.

XVIII. *In a case showing symptoms of focal infection, to what extent would you go in trying to save a non-vital tooth that apparently had no apical infection, provided such a tooth was a valuable factor in the reconstruction of the mouth?*

There is a certain range of opinions concerning the answer to this question. It runs from a purpose to save such teeth to a decision to extract them. Some recommend that such teeth be opened, cleansed, sterilized, and that the canals be sealed. Some open through buccal or labial plate and resect the root tip. Several advise elimination of all other foci before removing such teeth. The value of such teeth to the patient is referred to, on one hand; on the other, the probability of the good that removal of such teeth will do the patient is mentioned. The point of "putting it up to the patient" is also raised. Many express the opinion that, in serious cases, retention of such teeth is a somewhat "risky" procedure that is fraught, in some instances, with grave responsibilities. The answers show that no single rule for the handling of such cases can be adopted.

XIX. *In case a radiogram shows a tooth with a pulp chamber and portions of one or more roots only partly filled, how do you determine whether the unfilled apical portions of the roots contain vital pulp-tissue,*

or non-vital tissue, or no pulp-tissue at all—all three conditions being supposedly free from apical infection?

Most of the correspondents feel that there is no means, aside from exploring the canal with an instrument, of determining the vitality of apical pulp tissue. The efficacy of the electric current, in determining vitality or non-vitality of pulp tissue at the apex, is valued by some but considered of no value by others. Mention is made of close scrutiny of radiograms as an aid in determining effects on the pericemental membrane and the pericemental lamella (lamina dura) of the pulp tissue in the apex, in order to determine the probable condition of the pulp. Several show a desire to leave such teeth just as they are. Special mention is made of the fact that, after vital-pulp tissue was left in the apical portion, osteoblasts have closed the apical foramina.

XX. *In the reconstruction of a mouth, do you believe in removing non-vital teeth that do not show, radiographically, pathological conditions at the apices?*

This question brings out the real opinion of the correspondents regarding pulpless teeth that radiographically show no apical disease. Many are very decided in the view that they would not remove such teeth under the circumstances; others would be influenced by the usefulness of such teeth in the reconstruction plan; others again prefer not to include pulpless teeth in the reconstruction of the mouth, as they consider them rather "uncertain."

XXI. *Do you believe in root resection as a permanent operation? On which teeth? Do you feel that, in such an operation, the pulp-canal and dentin should receive special attention? Of what should such treatment consist?*

There is an interesting and wide range of opinion in this relation. Some have no use at all for the operation. Probably the greater number, while having an apparent suspicion that it is hardly the correct thing (as they seem unable in some cases to reconcile themselves to the conditions after the operation), admit that they somewhat favor the operation. A point of particular interest is the fact that one correspondent favors it in certain cases, to avoid a break in an otherwise perfect arch. Those who favor the operation frequently mention it as being indicated in carefully selected cases. The need for care-

ful attention to the root canal is extensively discussed. The indicated range for the operation varies from the centrals to the third molars.

XXII. *Should a tooth that is abscessed be extracted during the active process of inflammation, or is such an operation as a routine procedure contraindicated?*

A majority favor removal of such teeth, though some are opposed to it. Curetting after removal of the tooth is approved by some and opposed by others. Mention is frequently made of the need for careful post-operative dressings.

XXIII. *For the extraction of a considerable number of teeth in a case of serious infection, how do you proceed? Do you remove a few at a time or all at one operation?*

Most of the correspondents favor removal of a small number of teeth at a time. Among those who favor extensive removal of teeth, no. 23 refers to an interesting incident in his practice.

XXIV. *Do you consider that unerupted and impacted teeth should be removed, regardless of their location, when they cannot be brought into their proper places in the arch?*

While some favor removal of such teeth, on account of the trouble they may cause, a majority prefer to be governed by conditions affecting the individual case at the time a decision is made.

XXV. *Under what conditions, and in what locations, in the mouth, is it justifiable to remove a decomposed pulp and to fill the canal or canals? Give a list of such conditions.*

The patient's physical condition and the accessibility of root canals appear to be governing factors in the minds of most of the correspondents. Many are disinclined to endeavor to save a tooth if there is any destruction of its apical tissue. Mention is made of the fact that although a portion of the pulp may be decomposed, the apical portion may remain vital. Certain replies indicate a desire to eliminate *all* teeth in this condition; others show a desire to have the patient share the responsibility of retaining them.

XXVI. *Under what conditions, and in what locations in the mouth, is it justifiable to remove a vital pulp and to fill the root canals with an artificial substitute? Give a list of such conditions.*

From the answers to this question it is apparent that most of the correspondents favor preservation of the vitality of the pulp as long

as possible. Some consider that such conditions as exposures, pulp nodules, and secondary dentin, and the apparent early death of the pulp, justify its removal. An occasional correspondent would remove a pulp for purposes of reconstruction. Mention is made of contraindication in mouths in which there is much oral sepsis. The patient's health is also mentioned as a factor in determining whether a vital pulp should be removed.

XXVII. *When a tooth, to be extracted, has an apical infection, should the infected region be curetted after the extraction, or should it be left undisturbed? Would your decision to curette be the same if the granuloma came out with the root?*

A majority favor the curettement of infected sockets, but several express the opinion that it has been overdone. Opinions are divided as to whether or not a socket should be curetted when the granuloma adheres to the root of the extracted tooth.

XXVIII. *Upon what basis do you decide what to do with the various teeth in a mouth that contains a great number of non-vital teeth; some showing apical involvement and some not; some with partially and others with completely filled roots; some with exposed vital pulps and some with dead pulps? (Go sufficiently into detail so that your answer may serve as a guide in general practice.)*

There is almost a predominant opinion that the patient's health should govern the procedure in such cases. The importance of a carefully made oral examination, including full-mouth radiograms, study casts, vitality tests, and a careful clinical examination of the mouth, is referred to by several. Frequent mention is made of the desirability of removing all teeth that show apical infection. Reference is also made to the risk assumed in including questionable teeth in large reconstructions. Most of the answers show a desire to handle each tooth and each case with individual consideration. There is apparently no agreement on a plan of treatment for all cases of this type.

XXIX. *In the case of a maxillary-sinus infection that did not respond to treatment, what disposition would you make of any non-vital molars or bicuspids on the side affected?*

A majority favor removal of such teeth.

XXX. *In the reconstruction of a mouth, would you advise the removal of one or more vital pulps, if that procedure enabled you to use a more satisfactory type of bridge than would be possible if such pulps were not removed? If you ever advise such a procedure, does the patient's age and condition influence your decision?*

Opinions on this subject are divided, with those opposed to such a procedure in a majority. There is quite a range of opinion as to the effect of age on such a procedure.

XXXI. *From a histological standpoint, is the removal of a tooth pulp and the sealing of the canal a feasible operation? If not, why not? If so, under what conditions?*

Most of the correspondents feel that from a *practical* standpoint the operation may be satisfactory, but a much smaller number feel satisfied with it from a *histological* point of view. Of the latter, six give a very interesting explanation of what takes place in the apical portion of a root when a small portion of vital pulp is left uninjured. Several hold a somewhat different view on this particular point.

XXXII. *Do you believe in ionization? Why? To what extent?*

While some feel that "ionization" is a real aid in their practice, a majority are not very enthusiastic over it.

XXXIII. *In case a vital pulp has been removed, and a radiogram shows that the end of the root was reached and a slight apical injury probably resulted, due to the severing of the pulp tissue, do you feel that the result of the operation will be influenced in any way by the presence of mouth infection, pyorrhea alveolaris, or apical abscesses? What do you do in such cases?*

While a variety of opinions are expressed on this question, many believe, on general principles, that mouth infection, gingival and apical, should be eradicated before such an operation is undertaken.

XXXIV. *In the case of a patient who fractures the two upper central incisors, what is your procedure (a) when the pulps are exposed; (b) when they are not exposed but you fear they will die? To what extent does the patient's age influence your decision?*

The desirability of saving the pulp, if at all possible, is emphasized by most of the correspondents. However, when there is a condition as in (a), most of the replies indicate that the pulps should be removed and the roots filled. There are some exceptions to this, however.

In case of (b), need for the most careful supervision of the case is emphasized, particularly in younger patients.

XXXV. *In case the dentin of a non-vital tooth is not infected, and is surrounded by or encased in living cementum, which in turn is surrounded and nourished by a normal vital periodontal membrane, would you consider such a tooth a foreign body? Will it become infected, and will nature try to throw it off?*

Such teeth, in the opinion of practically all the correspondents, are not foreign bodies and nature will not attempt to throw them off. (See no. 18 for a slightly different view.)

XXXVI. *Do you believe in treating diseased apical tissue through the canal, or do you believe in surgical removal of such tissue?*

Opinions are divided on this question, but many feel that, when such conditions are treated through the canal, it should be in the simpler cases only. Ionization is referred to, but, for extensive apical involvement, the majority favor surgery. The patient's physical condition as a factor is mentioned.

XXXVII. *Do you consider that a diseased tooth with infected dentin is more liable to have a deleterious effect on the surrounding tissues, even though the canal was cleansed of the infected pulp, than would be the case if the symptoms of inflammation of the tooth pulp were detected early and the pulp removed before it had become infected?*

There is very little difference of opinion on this subject. The tooth with infected pulp removed "runs a poor second."

XXXVIII. *To what extent do you consider x-ray evidence final in determining the occurrence, extent, and nature of apical pathological conditions?*

The answers to this question are practically unanimous. They show quite clearly that while the radiogram is an extremely valuable aid in diagnosis, it is still to be considered *solely as an aid* and cannot be accepted as always showing indisputable evidence.

XXXIX. *In orthodontic treatment, what causes pulp death during the process of moving the teeth and how can that cause or causes be eliminated? What do you do or advise when pulp death occurs for this reason?*

Most of the correspondents concur in the opinion that pulp death in such cases occurs as a result of either too rapid movement of a tooth, or excessive stress upon a tooth in the closure of the jaws due

to change in position of the tooth. Either of these conditions causing an injury to the apical tissues, the circulation to the tooth is interfered with and pulp death results. Most of the correspondents recommend the saving of such a tooth, but the question of the development of the root is raised by some and its extraction advised.

XL. *Do you think one should differentiate between teeth that contain infected pulps and teeth from which healthy pulps have been removed, in determining whether or not non-vital teeth may be retained?*

Some feel that no distinction should be made, but a majority favor the tooth from which a healthy pulp had been removed. One correspondent raises the point that, in a sick patient, no distinction whatever should be made and that the pulpless teeth should be removed.

XLI. (*a*) *In correcting a case of malocclusion, do you first make a complete x-ray examination and vitality test of all the teeth in order to locate the unerupted or non-vital teeth?* (*b*) *If you make an examination, partial or complete, what do you do with the non-vital teeth present?* (*c*) *Do you complete your realignment with them in place or do you advise their early extraction?* (*d*) *What do you advise in case the third molars are impacted?* (*e*) *What do you think of the alveolus surrounding non-vital teeth as compared with that surrounding vital teeth?*

Opinions on the various points raised in this question vary considerably. A review of each answer is necessary.

XLII. *In your opinion, is dentin infected by preparing cavities for fillings and then allowing some time to elapse before placing the filling, no precaution being taken to seal the cavity in the interim and to prevent admission to it of saliva?*

While a few feel that dentin under such conditions is not infected, a majority believe that it is and that precautions should be taken to protect it.

XLIII. *May a non-vital tooth be free from infection?*

There is a division of opinions on this subject, but the view predominates that such a tooth may be free from infection.

XLIV. *May a vital tooth have an infected periodontal membrane (aside from that of a pyorrheal condition)?*

A majority believe that such a condition can easily exist. There is an interesting array of probable causes for such a condition.

XLV. *Does an infection at a root end come (a) from the tooth itself (b) through the canal (c) by the presence of infected dentine; or (d) is it carried there from the blood stream independently of infection in the canal? In case you consider that an apical infection comes from the tooth itself or the canal, how do you account for (e) the presence of infection in canals and at apices that have undamaged natural crowns? (f) If you consider that such an infection comes from the blood stream, why is it that, in a given mouth, some non-vital teeth are not infected and others, adjacent, may be badly infected?*

Most of the correspondents believe that root-end infection comes through the canal, but several express the belief that it may arrive by way of the blood stream or from the gingival crevice.

XLVI. *Do you believe that the presence of silicate fillings causes pulp death? If so, how do you prevent such action?*

There is a decided difference of opinion as to whether or not silicate fillings cause pulp death. A great many favor the use of some kind of a base or cavity lining for the purpose of protecting the pulp.

XLVII. *If you take the stand that all pulpless teeth, regardless of radiographic findings and regardless of the patient's health (good or poor), should be extracted, do you put any age limit on such procedure? Why?*

Opinions are practically unanimous in opposing such a stand.

XLVIII. *Do you consider that the manner of handling hospital cases, relative to problems of pulpless teeth, should govern the management of patients in private practice?*

There seems to have been a little misunderstanding of the point in this question. Nevertheless, there appears to be a difference of opinion whether a patient in private practice should have his pulpless teeth handled as they generally are in a hospital.

XLIX. *What do you think of the extraction of pulpless teeth, that radiographically show no infection, in patients in good health?*

Opinions are practically unanimous in condemning such practice.

L. *How do you view the construction of extensive restorations that call for the use of pulpless teeth as abutments?*

Some of the answers indicate willingness to utilize such teeth as abutments in reconstructive operations, providing radiograms show no apical-tissue destruction. A majority, however, are opposed to such practice. The answers to this question show some very interesting views on different types of bridgework.

LI. *What is your opinion on the use of large stationary bridges cemented to vital-tooth abutments?*

Interesting differences of opinion were expressed. A majority are opposed to the use of this type of bridge.

LII. *In case you had a pulpless tooth and a vital tooth proximating, with the pulpless tooth apparently free from infection and with a space adjacent to the pulpless tooth due to loss of teeth: Would you remove the pulpless tooth and carry the bridge to the vital tooth, or not?*

As a general rule, the correspondents show a desire to use the pulpless tooth. The patient's health and the quality of the root-canal operation are mentioned as factors of influence on the decision to save or extract.

III. REFERENCE TO THE QUESTIONS RAISED BY CORRESPONDENTS IN RESPONSE TO THE GENERAL INVITATION, TO THEM, TO "SUGGEST ANY OTHER QUESTIONS YOU THINK SHOULD BE ANSWERED."

Answers to the questions raised by the correspondents (page 295) will not be included in this work. It had been the author's expectation to publish a large number of answers to each question. This purpose required submission of each question to each correspondent. Execution of this plan would delay publication of the work to such an extent, however, judging from the experience in obtaining answers to the first set of questions, that the inevitable postponement would be unfair to those who have unselfishly given their time to the effort to make this study a success.

IV. CONCLUDING REMARKS

In conclusion, the author expresses the hope that this compilation will very materially help to solve some of the problems that have been disturbing the minds of the members of the dental profession.

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NOTES ON THE MINUTE OSTEOLOGY OF THE JAWS

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I. METHODS AND PROCEDURE

Some years ago, at the suggestion of Prof. Frank E. Blaisdell of the Medical Department of Leland Stanford Jr. University, the author undertook a study of the network of osseous trabeculae composing the spongy tissue of the mandibular and maxillary bones of man.

Cleaned and dried specimens were used. These were placed in containers of melted paraffin. The paraffin was stabilized for several hours in a fluid condition by inserting the containers in hot water. After a specimen was thoroughly impregnated with melted paraffin, the container was taken out of the hot water and allowed to stand. On the following day the hard paraffin was cut away from the surface of the bone. The bone was then placed in a vice and sectioned with a fine bracket saw. During the cutting, paraffin oil was used as a lubricant to prevent friction-heat from melting the paraffin and destroying the support for the delicate osseous reticulum of the spongy bone.

The sections of bone were then assembled and held in their proper positions by a loop of string, so that the parts could be fitted together when the sectioning was completed. The loop, with the sections in their proper order, was then inserted in xylol and left for a few days.

A second xylol bath was next used for a few days longer, in order to remove all traces of paraffin from the bone. The specimen was then allowed to dry.

II. REGIONS OF LOCALIZED EBURNATION ARE COMMON

Inspection and study of the dry specimens showed that regions of localized eburnation were common. Such calcified regions were associated with clear evidence of sepsis around the root ends of dead teeth, or with gingival suppurations. The loss of the alveolar crest around the necks of teeth, and the formation of a trough in its place, indicated gingival detachment and suppuration during life. Regions of destroyed bone (abscess) at the root ends of dead teeth in all cases showed some eburnation around the destroyed regions.

III. CALCIFICATION DIFFERENTIATED FROM OSSIFICATION

Regions of eburnation were regarded as osteo-sclerosis. There was a clear deviation from the appearance of normal cancellous bone. The spaces between the delicate trabeculae were found to be filled with what resembled the macroscopic appearance of cortical bone. This was referred to as sclerosed, since the blood supply could not have been normal in it.

The terms *eburnation*, *calcification*, and *osteo-sclerosis* are perhaps not generally understood. These terms may be correctly applied to the calcified areas described above.[1] Some men hold that *eburnation*, which is a sequela of osteo-sclerosis, is rare; but dental roentgenograms portraying this condition are very common.

IV. ROENTGENOGRAPHIC DISTINCTIONS

The osteology of the jaws would offer fewer difficulties if examinations of dry bones were made more generally. Such examinations would show that shadows commonly seen in dental roentgenograms should be interpreted as osteo-sclerosis or eburnation rather than as

[1] Calcification may be differentiated from ossification by the absence of structure. Ossification may be pathologic but the normal structure of bone will be in evidence. Calcification may be thought of as eburnation or a filling in of calcic debris.

hypertrophy. The latter term refers to a change in the shape or size of a part, while osteo-sclerosis or eburnation or calcification refers to a change not in size but in density. The observer of dry specimens would readily see the inappropriateness of the term hypertrophy, when applied to regions of eburnation.

The etiology of hypertrophy is not necessarily concerned with infection, while the etiology of localized osteo-sclerosis and eburnation in the jaws is concerned primarily with infection. A radiographic diagnosis that describes an eburnated area around an abscess as a *region of radiopacity on the edges of a radiolucent area*, indicating a *hypertrophy*, and terms the abscessed mass a *pericementoma*, seems to be inaccurate and confusing.

V. CAUSES AND SIGNIFICANCE OF EBURNATION

The eburnation commonly seen in the dry specimens of bone from the jaws is due to local and not systemic causes. In a study of the phalanges, some years ago, I observed that eburnated areas were not in evidence. If irritants in the blood stream, of systemic origin, were responsible for the eburnation commonly seen in the jaws, we should expect to find similar eburnation in other bones as well. Here attention should be called to the frequency with which eburnation is encountered in the mastoid process of the temporal bone. This calcification is commonly attributed to systemic irritants, especially luetic infections, in the blood stream. If this assumption were correct, eburnation would be found in bones that were not close to chronic centers of irritation, such as infected teeth or ears. Eburnation of bone is common in regions close to chronic infection but it is rare in regions not close to chronic infection.

VI. SIGNIFICANCE OF FIBROUS MARROW

Previous work by the author, in collaboration with Prof. Blaisdell, has shown the presence of fibrous marrow under all dead teeth.[2] The presence of fibrous marrow under such conditions usually indicates not a transient but a permanent irritation—an irritation due *to sepsis*

[2] Novitzky, with the collaboration of Blaisdell: *Journal of Dental Research*, 1922, iv, p. 299; Thoma: *Ibid.*, p. 321; Novitzky: *Ibid.*, p. 323.

rather than to chemicals used in the "treatment" of dead teeth. Chemical irritation in the absence of infection is but temporary. Fibrous marrow appears in both acute and chronic conditions involving marrow, and is due to the temporary or permanent irritation caused by trauma or infection and reinfection. Localized fibrous marrow is an indication of nature's effort to wall off and isolate injurious agents; persistent bacterial infection resulting in localized osteo-sclerosis. In acute inflammatory conditions involving marrow, the fibrous marrow is less in amount and soon gives way as resolution takes place; in subacute conditions, it is more abundant and more persistent, and markedly so in chronic conditions. In all cases it probably disappears when all sources of infection or irritation are removed.

VII. CALCIFICATION RESULTING FROM AN INFLAMMATORY REACTION TO A LOW-GRADE INFECTION IS A PATHOLOGICAL PROCESS

Eburnation may be regarded as a calcification—a process mainly retrograde and persistent as regards time—the result of a condensing osteitis carried to an extreme degree (pathological). It should be emphasized that calcification, resulting from an inflammatory reaction to a low-grade infection, is not a physiologic ossification but a pathologic calcification. In the healing of fractures, a physiologic ossified callus is not expected by a surgeon until all inflammatory symptoms subside; and in compound fractures, not until all infection and suppuration have run their course and all necrotic tissue has been thrown off. When a dead infected root has been amputated, the calcification that takes place in the bone-wound around the stump is not a physiologic ossification. It is pathologic. If the gum-wound closes over the bone-wound, x-ray study will in time show areas of "radiolucence." These areas are often pointed to as evidence of "cures," of regeneration of bone. But operation on such regions reveals odor and offensive granulation tissue; bacteriologic cultures show streptococcal and other infecting microörganisms; and microscopic examination of the tissue shows evidence of changes in structure warranting a diagnosis of osteo-myelitis.

If the tissues do not close over the amputated root end and it drains through the scar tissue into the mouth, the bone around the

edges of the operative wound becomes normal; a thin edge only takes on the appearance of a thin plate of cortical bone.

VIII. REMARKS ON GRANULOMATA AND ON ROOT-CANAL FILLING

If there is a fistula to the mouth, from the root ends of an abscessed tooth, nature's inflammatory blood solids may serve to lessen absorption by way of the blood stream in the marrow spaces. Dentists are familiar with the discharge that takes place every few weeks from a "gum boil." As the volume of fluid and gas pressure increase in the abscess, a point is reached at which there occurs a discharge through the fistula into the mouth.

If the gas generates when there is no fistula, it will travel in the direction of least resistance. This may be between two cortical plates of bone with no vent under the periosteum, or it may be through a thin layer of bone under the antral membrane. In the bone, there is developed a suppurative cavity, which may contain exuberant granulation tissue, gas, and streptococcal fluids. The offensive contents of such a cavity are commonly called a granuloma. Strange to say, a granuloma has been regarded by some dentists as harmless—by one of them as an *organ* beneficial to the body.

Glib talk about harmless granulomata, and x-ray pictures in which they are misinterpreted as young areas of new bone (!), have done much to maintain the dangerous practice of tooth-pulp devitalization. Every surgeon knows, and every dentist should know, what would happen if one should rip out the contents of the inferior dental canal of the mandible, plug this canal with rubber to its most minute cribriform opening, and allow a half inch of the plug to stick into the mouth so that the tissues did not cover it. There would be a gateway for the constant entrance of infection and reinfection in such measure as to be capable of causing death. But the procedure in "removing the nerve of a tooth," and sealing the canal, is even worse, for, in the first case, all blood vessels leading to the mandible would not be destroyed.

A surgeon would not expect a bone implantation, aiming to bridge a space in the mandible, to succeed if one end of the implant were allowed to stick into the mouth. The result would inevitably be suppuration and loss of the implant and neighboring tissue.

The body does not rid itself of a dead tooth as it rids itself of an infected bone implant, because the dead tooth is ankylosed by a deposit of cementum. If cementoblasts did not lay down lacunated cementum on the roots of dead teeth, the body would rid itself of such teeth just as it rids itself of an infected bone implant. This is true not only of the dead tooth that is vile and green from decomposition, but also of the dead tooth that has not reached so advanced a stage.

IX. GENERAL CONCLUSIONS

Every careful study in the field of the osteology of the jaws reinforces the fact that every dead tooth is infected; that no method of "treatment" has succeeded in rendering a dead tooth permanently sterile and harmless; and that the only safe procedure, in the case of a dead tooth, is surgical removal of the tooth and surrounding regions of infection.

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STUDIES ON THE RELATIONS OF TYPES OF DIET TO DENTAL CARIES

A Statistical Survey of Children under School Age Relating to two Dietetic Theories on the Etiology of Dental Caries

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I. INTRODUCTION

The investigation herein reported was undertaken to determine whether or not certain theories of the dietetic causation of dental caries are supported by actual findings. Such theories have frequently led to the unproved contention that desirable results may be achieved by the modification of a supposedly pernicious regimen in the national dietary. Usually, these general statements have been brought forward in uncritical anticipation of such changes; so far, however, as the literature of the subject reveals, the hypothetical results of such changes have not been verified by sufficient observation.

Of the many theories which have invited attention, only two of the best known are the subject of this investigation. The first is the

"*physiological*" *oral hygiene* thesis as defined by Wallace (9), which may be summarized as follows: *Caries is initiated by acid fermentation of sticky foods which adhere to the teeth. Cleaning the teeth after meals prevents such fermentation. The same result is effected by mastication of "natural" foodstuffs which are detergent, and preferably acid in reaction, at the termination of each meal. "Physiological" oral hygiene so maintained will prevent dental caries.* This study seeks to determine whether or not this thesis is substantiated by a careful statistical survey.

The second theory examined is very popular: *Indulgence in sugars leads to dental decay.* Whether or not a minimal use of sugar is found to lower the incidence of dental caries is the second object of this inquiry.

This study has been made on children of pre-school age, namely, under six years. There are three advantages in studying this period: it is an age of mass incidence of dental caries; it is the age of relatively simple dietaries; it is the age when dietaries are fairly well ascertainable. In addition, Kansas City, Missouri, where this survey was made, offers an opportunity for study in children of this age not found elsewhere in the country. It has been through the fullest coöperation of its Children's Bureau, a publicly supported civic institution, that the conduct of the study was possible. This is the only city that has been wise enough to continue, on its own resources, the admirable physical survey of children of pre-school age first established by the national government in all parts of the country during the war.

The particular theses will be detailed later. They have been selected not only because they are representative of current thought on this subject, but because they are the most susceptible of investigation by the statistical method.

II. METHOD OF STUDY

The examinations were made in good daylight in the course of complete physical examinations by well-trained medical men. Only a few were asked to coöperate in this study, and each one showed interest and thoroughness. The examiners bared the teeth with ordinary tongue blades. Each tooth was observed separately, and the

findings dictated to a secretary, who recorded them on cards (*fig. 1*). Many of the mouths were checked by the writer, who found that the examinations were made and recorded accurately. In some doubtful cases the writer improvised probes of splinters from the tongue blades for corroborative instrumentation.

It is definitely admitted that the method is not above the criticism that dental instrumentation was not used; the incidence of the caries found should doubtless have been greater. However, a collateral

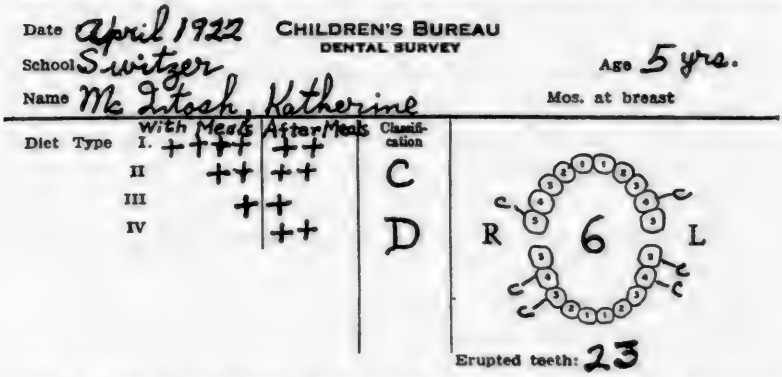

FIG. 1. TYPE OF CARD USED IN THE COLLECTION AND CLASSIFICATION OF DATA

study, by the Children's Department at the Deaner Institute, showed an error by this method in only 8 per cent of the number of mouths examined and in only 0.5 per cent of the number of teeth. The opinion of a dentist (5), in constant observation of children's mouths, was expressed as follows:

"In general, any cavity in a deciduous tooth, detectable by a dentist only, will become distinctly obvious to the untrained eye before another two months passes, if it is not treated."

This report, then, relates to gross caries, obvious to careful direct inspection without dental instrumentation.

Following the physical examination and the dressing of the children, the child and the mother came to the writer, who inquired in detail from the mother into the child's dietary. The clinical analysis was made as the mother talked, and the entire dietary for that child was

classified before the next case was taken up. Somewhat over an average of five minutes was given to each mother.

All interpretations have been made conservatively in order to strengthen their validity. Six classifications have been made. *Class A* consists of the cases of children on physiologically unhygienic diets. It is aimed to include in class A only diets of distinctly unhygienic nature, as defined from the point of view of Wallace. Thus, a child who eats a cracker or a cookie two or three times a day, never chewing on an apple, orange, or the like between meals, would come into class A. Had he eaten only one apple a day, he would come out of class A and fall into class C, the indifferent group.

Class B, the hygienic group, consists of those children who daily eat detergent food between or after meals, and who do not even once a day use anything which might stick to the teeth and there undergo acid fermentation. The addition of a cracker a day, or of a piece of candy, or the like, to the diet of these children would put them into class C, regardless of the number of apples, oranges, or pineapples consumed daily between meals.

Class C, the indifferent group, comprises the great majority of children. It is intermediate between classes A and B.

Class D includes children that use excesses of sweets, or (as estimated) more than fifty pounds of sugar a year.

Class E consists of children that use a minimum of sweets, or (as estimated) less than sixteen pounds of sugar a year.

Class O is made up of children that use sweets in moderation; that is (as estimated), between sixteen and fifty pounds of sugar a year.

III. USE AND ARRANGEMENT OF DATA

The data, after collection and analysis as described, were separated into age groups for each school district. A cumulative report covers the first twenty school districts, representing 1,068 cases. All data are represented on the accompanying tables and graphs. In each table, the upper figure in a pair shows the number of mouths actually represented in the group; the lower figure gives the percentage represented by the number immediately above it in the corresponding total. The percentage in parenthesis, in each instance, represents the ratio of a corresponding total above it to the total number for the

year, or other corresponding group, which may be easily inferred in any case.

In this study a carious mouth is a mouth presenting one carious area, or more, obvious enough for detection by direct inspection without instrumentation, as before described.

A previous report (8) detailed the findings relative to the incidence of dental caries, regardless of diet, in these young children.

The findings have been arranged by ages of the children studied. Although dental caries is not wholly absent in the tots less than two years of age (four cases), the incidence rises sharply at the 3–4 year period and is high thereafter. For this study, therefore, the 3–6 year period may be considered the age of the mass incidence of dental caries for the deciduous teeth, and has been designated the "caries period." For this reason all the data of these three years are given collectively and are summarized in each report. The most significant of the data presented should and do fall within this period, as we shall see.

IV. "PHYSIOLOGICAL" ORAL HYGIENE AND DENTAL CARIES

Interesting speculations, by various thinkers, have evolved numerous logical and enticing theses regarding dietetic causation of dental caries. Several are the product of reasoning from physical, chemical, and microscopic data that are both valid and widely known. Most of the data relate to the work of such authorities as Pickerill (7), Miller (6), Williams (11), and Gies (3). They have done fundamental work.

In the main, these theses may be represented by a quotation from a government bulletin on "Child care" (10):

> "Dental decay results from the acids produced by the fermentation of food particles remaining in the mouth after eating. Soft, sweet, sticky, and pasty foods fill the recesses between the teeth or any irregularities, where they readily ferment. For this reason, when foods rich in starches and sugars have been eaten alone, or at the close of a meal, it is most important to scour the teeth and rinse the mouth with especial care. The daily use of some hard foods, such as raw fruits like apples or pineapples, celery, toast, and other wholesome but resistant foods, aids in keeping the teeth and mouth clean."

Wallace (9) has given as detailed, as emphatic, and as decisive a treatment of this subject as any writer. In consequence, his particular thesis of "physiological" oral hygiene has been selected as an object of this inquiry. In section VII of this paper, his point of view will be adequately presented by extensive quotations from his work, "Child welfare." Postponed to that section also is the detailed analysis of the cases observed and the discussion of thelogic of the statistical method employed.

Close examination of the figures, in the cumulative report of the 1,068 cases, gives no findings that definitely support the thesis of "physiological" oral hygiene of Wallace, apparent support given it by figures for the last three years of pre-school age notwithstanding. The findings in the "end-results period"—five to six years of age—strongly suggest that the conclusions of Wallace are not confirmed by actual experience. This is warranted, so far, at least, from the present critical study of a clinical-statistical nature. Children of that last age, who have been on such diets as those advocated by Wallace, present no other results for caries than do their fellows of the same age who live on indifferent diets, or than those living on diets decidedly unhygienic as defined by the thesis under study.

V. SWEETS AND DENTAL CARIES

That present-day indulgence in sweetmeats is a factor that is largely responsible for the high incidence of dental caries in civilized communities is a thesis now so commonplace that it has well-nigh become a dictum with authority. Kirk (4) says:

". . . The evidence is almost overwhelming that the inordinate and increasing habitual use of sweets by civilized children is a custom pernicious alike to the integrity of their dentures and to their general health."

Fones has written (1):

"When we consider that dental caries can only be produced from starch and sugar, and that the starch must be converted to dextrose before it can be converted into lactic acid, it is quite truthful to make the statement that all dental decay is produced by sugar. In the Bridgeport schools we are using this truism in the following jingle:

'Children you should learn the truth;
Nothing but sugar decays a tooth.' "

The details of this study of sweets and dental caries are presented in section VII. Briefly stated, they indicate almost a complete reversal of the findings to be expected, if a minimal use of sugar prevented caries and a maximal use of sugar induced it.

VI. SWEETS PLUS "PHYSIOLOGICAL" ORAL HYGIENE AND DENTAL CARIES

In view of these adverse findings, it seems well to determine whether a combination of "physiological" oral hygiene plus a minimal use of sweets will give other results. Analysis of the data, as will be shown in section VII, gives none. No protection from caries was found in the group of children whose diet was the combination supposedly most advantageous; "physiological" oral hygiene plus a minimal use of sweets had no advantage, so far as protection from dental caries was concerned, over the opposite extreme of dietary.

VII. DISCUSSION: DETAILED ANALYSIS OF CASES

Discussion of the methods, theories, and analysis of the figures has been brought together in this section for critical study.

i. The Logic of the Statistical Method

This is a statistical report. Statistical inquiry is of definite value, if the logic of the method is not abused. It is definitely understood that proof or disproof, of the absolute validity of hypotheses which relate to dental caries, cannot be established by statistical methods alone. Notwithstanding this, it is believed that critical consideration of such theories may advantageously be based upon a carefully controlled statistical survey of the effects of their application. The justification of these theories is materially strengthened, or weakened, as data from a large number of cases are found definitely to bear out, or not to bear out, the predicted advantages.

The larger the group investigated the greater the validity of the deductions. The interpretation of a large mass of data will be but slightly influenced by limited additions. It is understood, then, that this report, based on only 1,068 cases, must be looked upon as a preliminary statement. Although these thousand-odd cases may

reasonably be considered representative, investigation of five or ten thousand more cases is desirable.

ii. Use of an Arbitrary Food Classification

With special reference to the thesis of Wallace, the foods have been divided into four types, as follows:

Type I. Soft sticky foods: as breads of finely ground flours; cookies, pies, cakes, crackers, etc.; pancakes, waffles, doughnuts; noodles, spaghetti, macaroni; mashed foods, such as potatoes or vegetables; starchy foods, such as dry or steamed cereals; nuts.

Type II. Sweet sticky foods: as candies, sugars, jams, preserves, syrups, sorghum, honey; bananas, figs, dates; ice cream.

Type III. Chewable detergent foods: as fruit pulps, apples, peaches, pears, prunes, etc. (figs, dates, raisins, and bananas excepted); leafy and stalky vegetables (not mashed); breads of coarsely ground whole flours; whole meats and fish; chewing gum.

Type IV. Sour detergent foods: as acid fruits or fruit juices, such as oranges, grapefruit, pineapples.

Particular note was made of the time the foods were used in relation to meals. Those used during the meals are represented by + signs to the left of the vertical line on the card (*fig. 1*); foods used at the end of a meal or between meals ("piecing" between meals, as the mothers term it) are represented by + signs to the right of the line. Clinical quantitation is represented on the four-plus basis, a convenient device widely used in clinical medicine. In this study a piece of bread, cookie, or the like, eaten less frequently than every day between meals, would be represented by + for diet-type I to the right of the corresponding line (*fig. 1*). If it were eaten daily, it would be represented by ++. If it were eaten twice, almost every day, it would receive +++. If more than twice every day, it would be marked ++++. Likewise, in diet-types III and IV, ++ would represent a raw apple or its equivalent eaten every day. Less would be indicated by +; more, by +++; two or more apples every day, by ++++.

In diet-type II, + represents not more than a level teaspoonful of sugar or its equivalent three times a day. By actual measurement this is less than sixteen pounds of sugar a year. Three heaping

teaspoonfuls a day, or the equivalent (about fifty pounds a year), is represented by ++. More is recorded as +++ or ++++, as the amounts indicate. The data thus recorded were then classified into groups, as shown below. (The horizontal lines indicate that any amounts of the corresponding food types may be used.)

Classification A. "Physiologically" unhygienic diets

I ———	+++ (or more)	I	+++ (or more)	———
II ———		II	———	———
III ———	Not over +	III	0	0
IV ———		IV	0	0

Classification B. "Physiologically" hygienic diets

I ———	Not over +	I	Not over +++	0
II ———		II		0
III ———	+++ (or more)	III	+++ (or more)	———
IV ———		IV		———

Classification C. "Physiologically" indifferent diets

Any other than in A or B

Classification D. Excess of sweets in diet

II +++ (or more). (Regardless of relation to meal time)

Classification E. Minimum of sweets in diet

II Not over + (Regardless of relation to meal time)

Classification O. Moderation in sweets in diet

II ++ (Regardless of relation to meal time)

iii. "Physiological" Oral Hygiene and Dental Caries

The theory under analysis is adequately represented by the following quotations from Wallace (9) that explain his dietetic philosophy.

". . . Between the age of two and two-and-a-half years, food of a still more detergent nature should be introduced into the dietary, so that each meal will terminate in such a way as to leave the mouth physiologically clean. To reduce this conception to a practical form, fresh fruit should be introduced into the dietary in such a way as to terminate each meal. . . . The type of meal, then, which is required for oral hygiene and the prevention of dental diseases should be fully developed at the end of the second, or at latest at the end of two-and-a-half years—that is to say, the bulk of the food should require and stimulate thorough mastication . . . and food of a detergent nature should terminate the meals" (p. 15).

TABLES I AND II "PHYSIOLOGICAL ORAL HYGIENE" AND ITS RELATION TO DENTAL CARIES

AGES BY YEARS		0 TO 1			1–2			2–3			3–4			4–5			5–6			AVERAGE 3–6			AVERAGE 0–6		
TOTAL MOUTHS BY YEARS		68 (6.4%)			212 (19.9%)			218 (20.4%)			203 (19.0%)			203 (19.0%)			164 (15.4%)			870 (82.5%)			1068 (100%)		
		TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS
A. ON "UNHYGIENIC DIETS"	#	3	0	3	20	1	19	23	5	18	21	7	14	16	6	10	35	25	10	74	44	30	120	69	51
	%	(4)	0	100	(9)	5	95	(11)	22	78	(10)	33	67	(9)	33	67	(21)	71.5	28.5	(13)	59.5	40.5	(11)	57.5	42.5
B. ON "HYGIENIC DIETS"	#	1	0	1	8	0	8	38	1	37	22	3	19	26	7	19	20	16	4	68	26	42	315	50	265
	%	(1)	0	100	(4)	0	100	(17)	2.6	97.4	(11)	14	86	(13)	27	73	(12)	80	20	(12)	38.2	61.8	(11)	15.9	84.1
C. ON INDIFFERENT DIETS	#	64	0	64	184	7	181	157	17	140	160	51	109	157	64	93	109	68	41	428	183	245	933	303	630
	%	(94)	0	100	(87)	3.8	96.2	(72)	11	89	(79)	32	68	(78)	40	60	(67)	62.4	37.6	(49)	42.7	57.3	(87)	32.5	67.5
TOTAL	#	0	0	0	4	1	3	23	5	18	61	7	54	83	12	71	101	75	26	253	44	209	701	126	575
	%	(0)	0	0	(2)	25	75	(11)	22	78	(30)	11	89	(41)	14.5	85.5	(62)	74.2	25.8	(44.4)	17.4	82.6	(65.6)	17.9	82.1
F. CARIOUS MOUTHS	#	0	0	0	2	1	1	5	1	4	7	3	4	12	7	5	25	16	9	44	26	18	95	50	45
	%	(0)	0	0	(50)	50	50	(22)	20	80	(64)	43	57	(59)	58.5	41.5	(75)	64	36	(59.5)	59	41	(57)	52.6	47.4
G. NONCARIOUS MOUTHS	#	68	3	65	208	19	189	195	18	177	142	19	123	120	19	101	84	52	32	209	18	191	806	76	730
	%	(100)	4	96	(98)	9	91	(89)	9	91	(70)	13	87	(59)	16	84	(50)	62	38	(50.5)	8.6	91.4	(75.5)	9.4	90.6
INDIFFERENT DIETS																									
HYGIENIC DIETS																									
UNHYGIENIC DIETS																									
TOTAL																									
INDIFFERENT DIETS																									
HYGIENIC DIETS																									
UNHYGIENIC DIETS																									
TOTAL																									
INDIFFERENT DIETS																									
HYGIENIC DIETS																									
UNHYGIENIC DIETS																									
TOTAL																									

FIG. 2. TABLES I AND II.[1] "PHYSIOLOGICAL" ORAL HYGIENE AND ITS RELATION TO DENTAL CARIES. SEE GRAPHS I AND II.

[1] In all the tables and graphs, the *upper* figures in a vertical pair represent the actual number of specific cases observed; the *lower* figures represent the *percentage* of the actual (upper) number of cases in the corresponding total number of cases.

". . . Food should be of such a nature or the meal so arranged that the mouth and teeth will be left physiologically clean at the end of the meal" (p. 37).

". . . Since the teeth of animals and primitive man are kept in a hygienic state by their foods, it is a simple guide to say that food in its natural condition will, if taken at the end of a meal, leave the mouth clean. For various reasons I have drawn attention to the value of uncooked fruit for this purpose . . ." (pp. 37–38).

"That the gustatory secretions should be stimulated at the end of a meal not by sweets, but by food of a natural, hygienic, or detergent nature, is evident to all who admit the principle that the mouth should be left physiologically clean. Now what does so most effectually? Fresh fruit certainly does: not only the dilute sugars, but the acids, the aromatic and even the acrid substance in the fruit do so most effectually. Fruit contains but little nutriment, and its real value is hygienic and not nutritive; much less is it a digestive tonic. . . . Fruit leaves the mouth in an hygienic state" (pp. 42–43).

". . . There is only one proper solution to the problem: primarily we must direct our immediate efforts to teaching the teachers of physiology the elements of the physiology of oral hygiene and try to bring home to them the stupendous havoc in the teeth and other parts which erroneous teaching or the absence of teaching has given rise to in the past. Then nurses, health visitors, medical men, and dentists will become able to teach harmoniously, and then the fundamental principles of physiology, although consciously unknown to the multitude, will establish customs, habits, and current knowledge which will wipe out one of the darkest and most widespread stains of our civilization" (p. 19).

Examining the cumulative report of 1,068 cases, the following points are revealed. The data referred to are found in tables I and II and are blocked out in percentage form on graphs I and II.

(1) In the "caries period," three to six years of age, (a) a moderate difference in the percentage incidence of caries is shown on the unhygienic and hygienic diets: 59.5 per cent and 38.2 per cent, respectively. There is 21.3 per cent less caries on the hygienic diets.

(b) Conversely, the incidence of immune mouths is 40.5 per cent on unhygienic diets and 61.8 per cent on hygienic diets, the same difference of 21.3 per cent existing to the advantage of the hygienic group.

(c) On unhygienic diets the cases with caries show a slight majority of 59.5 per cent. (This is 19 per cent more than the immune cases:

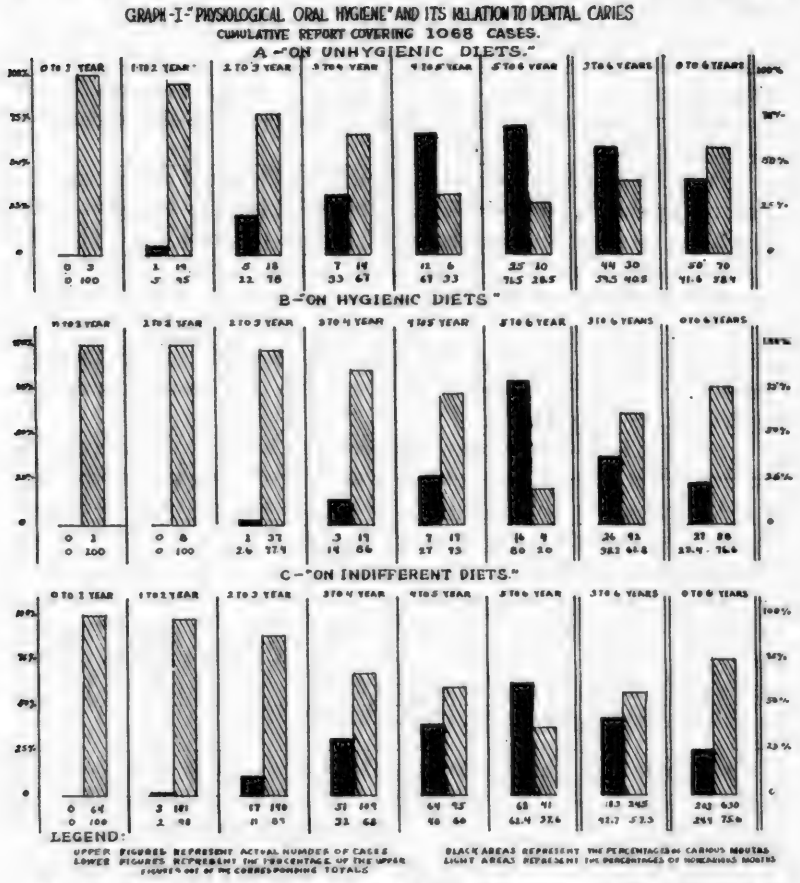
GRAPH-I-"PHYSIOLOGICAL ORAL HYGIENE" AND ITS RELATION TO DENTAL CARIES
CUMULATIVE REPORT COVERING 1068 CASES.
A-"ON UNHYGIENIC DIETS."
B-"ON HYGIENIC DIETS"
C-"ON INDIFFERENT DIETS."
0 TO 1 YEAR
1 TO 2 YEAR
2 TO 3 YEAR
3 TO 4 YEAR
4 TO 5 YEAR
5 TO 6 YEAR
3 TO 6 YEARS
0 TO 6 YEARS
LEGEND:
UPPER FIGURES REPRESENT ACTUAL NUMBER OF CASES
LOWER FIGURES REPRESENT THE PERCENTAGE OF THE UPPER FIGURES OUT OF THE CORRESPONDING TOTALS
BLACK AREAS REPRESENT THE PERCENTAGES OF CARIOUS MOUTHS
LIGHT AREAS REPRESENT THE PERCENTAGES OF NONCARIOUS MOUTHS

FIG. 3. GRAPH I.[2] (SEE TABLES I AND II.) "PHYSIOLOGICAL" ORAL HYGIENE AND ITS RELATION TO DENTAL CARIES.

Black areas represent the percentages of carious mouths. *Light* areas represent the percentages of non-carious mouths. *A*.—On "unhygienic" diets. *B*.—On "hygienic" diets. *C*.—On "indifferent" diets. The first six columns pertain to the successive six years; the seventh and eighth columns, to the 3–6 year and the 0–6 year periods, respectively. The mathematical data are taken from the tables I and II, where they may be studied to greater advantage.

[2] The author's graphs (six) were not properly prepared for satisfactory reproduction. The indistinct legend on each is reprinted at the bottom. The indistinct numerals on the graphs may be verified in the corresponding tables.—(Ed.)

40.5 per cent.) The cases on hygienic diets reverse the division—61.8 per cent are immune and only 38.2 per cent are carious—a difference of 23.6 per cent, again in favor of the hygienic diet.

(d) Similar proportions are found to uphold the thesis under study in the lines F and G. Of the cases showing caries, those on unhygienic diets are to those on hygienic diets (44:26) roughly as 5:3. It is about as 5:7 in the non-carious group of mouths (30:42 in actual cases).

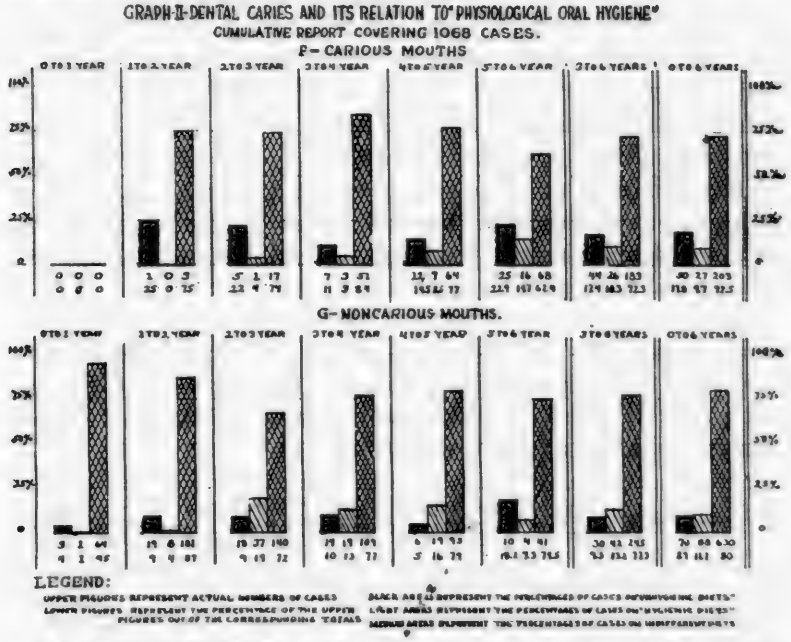
GRAPH II-DENTAL CARIES AND ITS RELATION TO "PHYSIOLOGICAL ORAL HYGIENE"
CUMULATIVE REPORT COVERING 1068 CASES.
F- CARIOUS MOUTHS
G- NONCARIOUS MOUTHS.
LEGEND:

FIG. 4. GRAPH II. (SEE TABLES I AND II.) DENTAL CARIES AND ITS RELATION TO "PHYSIOLOGICAL" ORAL HYGIENE.

Black areas represent the percentages of cases on "unhygienic" diets. *Light* areas represent the percentages of cases on "hygienic" diets. *Medium* areas represent the percentages of cases on "indifferent" diets. *F.*—Carious mouths. *G.*—Non-carious mouths. The first six columns pertain to the successive six years; the seventh and eighth columns to the 3–6 year and the 0–6 year periods, respectively. The mathematical data are taken from tables I and II, where they may be studied to greater advantage.

(e) The ratios for caries to non-caries are indicated below:

	AS SIMPLE RATIOS	IN ACTUAL CASES
In the whole age group	8:10	253:317
On unhygienic diets	8: 5	44: 30
On hygienic diets	8:13	26: 42
On indifferent diets	8:11	183:245

Thus, for the moment, it would seem that the thesis is upheld.

(f) There is, however, one point that should be noted here, since it comes from this 3–6 year period. Of children living on rigorously hygienic diets, as defined by the quotations, 38.2 per cent present mouths with obvious caries. This is better, indeed, than the 59.5 per cent on unhygienic diets; but it nonetheless materially weakens the validity of a thesis which would hold that (9, p. 87) "dental caries and consequent and concomitant diseases will soon practically cease to exist in all families which are brought up with reasonable care and intelligence," as heretofore defined.

(2) Considering now the last age unit—five to six years of age—we notice that, (a) for all cases seen (164), disregarding their diet groupings, the ratio between caries and immunity is 109:55, or simply as 2:1. This ratio is changed somewhat in the limited groups, as is shown in the appended summaries.

Caries: Non-caries ratios in the 5–6 year group

	AS SIMPLE RATIOS	IN ACTUAL CASES
For total cases	20:10	109:55
Unhygienic cases	25:10	25:10
Hygienic cases	40:10	16: 4
Indifferent cases	17:10	68:41

The ratio which is the most unfavorable chances to be in the hygienic group of cases.

(b) In the gross division of all the cases between the three diet groups, unhygienic (A), hygienic (B), and indifferent (C), we find the ratios to be A:B:C::21:12:67 (expressed in percentages of the total number of cases). This A:B:C triad is moderately changed when

partition is made into the carious and non-carious groups. (Cf. lines F and G.)

A: B: C ratios in the 5–6 year group

	AS SIMPLE RATIOS	IN ACTUAL CASES
For total cases	10:6:31	35:20:109
Carious mouths	10:6:27	25:16: 68
Non-carious mouths	10:4:41	10: 4: 41

The hygienic diets are not found associated preponderantly with non-caries. Indeed, the converse is seen to be the case—in the non-carious cases there are the very fewest on hygienic diets.

(c) In the listings under carious mouths, the Unhygienic:Hygienic ratio is approximately as 5:3 and the corresponding Unhygienic: Hygienic ratio in the non-carious mouths is 5:2 (25 and 16 cases on line F, and 10 and 4 cases on line G, respectively). Here again the smaller proportion of hygienic diets is found in the immune group of cases. It should also be noted that this ratio for all the cases in this age group (35:20) is practically as 5:3.

(d) Although in each observation some numerical disadvantage was seen against the cases on hygienic diets, not enough disproportion was present to be significant. For the method used, I look upon these ratios as fairly similar, the slight variations noted being of value only to prevent equivocal readings, such as might arise if the small advantages were on the opposite side of the neutral point. The figures, then, show a fairly constant incidence of caries or immunity regardless of the "physiological" oral hygiene of the diets.

From this, the point may well be taken that the tentative conclusions elicited from the 3–6 year column must be carefully questioned. That column was chosen arbitrarily as representing the "carious period." While this is not disputed, careful note must be taken that the percentage incidence of caries mounts rapidly in that period (30 per cent—41 per cent—67 per cent). Consequently, the 5–6 year group may adequately be judged representative of the end-results in that three-year period. Thus, any contrary findings early in the period (*i.e.*, 3–4 and 4–5 years), if included, will naturally invalidate conclusions rightly derived from the "end-results period," that is the 5–6 year group. From the point of view of this dental pathology

and its etiology, it will be readily conceded that, in the children about ready for schooling, the essential object of observation and discussion is the "end-result." There is little advantage in a low incidence of caries at three to four years of age, if this low incidence cannot be maintained to later years, even to the six-year period. Therefore, our period of conclusive deduction is that of the "end-results period," *i.e.*, in this study, five to six years of age.

iv. Sweets and Dental Caries

The current opinion on sweets is adequately presented by the four following quotations.

"When the diet of the average American child is considered, it is not so astonishing to find that dental caries is a universal malady. Originating as it does from carbohydrate fermentation upon the teeth, no other result could be expected among the children of a nation averaging nearly one hundred pounds of sugar per capita per year. The writer personally believes that in childhood, a well balanced diet, with special emphasis upon the calcium-content of foods, and the elimination of free sugar, would result in practical immunity from dental caries, even in the absence of a rigid system of mouth cleanliness. . . . It is reasonable to believe that susceptibility is mainly in proportion to the consumption of free sugar, and that immunity is acquired, in a great measure, by the absence of free sugar in the dietary. . . . Clinical experience and general observation seem to show, however, that the high consumption of starchy food, if unaccompanied by free sugar, does not result in dental decay. In fact the evidence is all against the sugar."—*Fones* (2).

"As regards sweets, all are good in that they are salivary stimulants, and all are bad in that they are readily fermentable; and as a general rule it may be said that the ill effects quite outweigh the good effect. They all (practically) leave sugar behind in the mouth to be transformed into lactic acid—chocolates and caramels most, and hard-boiled sweets like toffee least. The old-fashioned "acid drops" are possibly the least harmful."—*Pickerill* (7, p. 344).

"The two great pitfalls to avoid (in the feeding of children) are sweetening of the food and the giving of pap. Regarding the former there is an almost ineradicable belief that the craving for sugar is a natural instinct in a child. . . . It is an interesting fact that sugar with one exception (honey) always occurs in Nature mixed with a very large proportion of

acid salts and of detergent fibrous tissue, and it is in this harmless state only that the child should get his sugar—namely, as ripe uncooked fruit. If the sugar allowed the child were extracted by him from, say, sugarcane, he would have to perform a lot of work to get a small quantity, and therefore would not get an excess of it, and what he did get would be in a form that is absolutely harmless. What should be condemned is the giving of sugar in an unnatural, too concentrated form, such as jam, sweets, chocolates, cakes, etc., and especially at wrong times. It should always be remembered in connection with sugar that the craving for it is very easily acquired, and long before anyone is aware of it the child is getting an injurious amount. The addition of sugar to a sufficient diet of bread and potatoes is a case of carting coals to Newcastle!"—*Wallace* (9, pp. 68–69).

"Foods in the form of sweets, jam, rolls, bread and marmalade, chocolate, etc., are to be condemned absolutely as meal terminals, as their effect is the opposite of fruit, which leaves the mouth in an hygienic state."—*Wallace* (9, p. 43).

Close examination of the figures, in this study of sweets and caries in 1,068 cases, reveals the following points, all derived from tables and graphs III and IV.

1. In the "caries period" again, three to six years, (a) a moderate difference in the percentage incidence of caries is shown between cases using excess of sweets and a minimum of sweets (53.4 per cent and 32.4 per cent, respectively). There is 21 per cent less caries in those using a minimum of sweets.

(b) Conversely, between the immune cases on an excess of sweets and on a minimum of sweets (46.6 per cent and 67.6 per cent, respectively) there is a like difference of 21 per cent in favor of a minimal use of sweets.

(c) On excess of sweets, the cases with caries show a very slight majority of 53.4 per cent (only 6.8 per cent more than the immune cases). The cases on a minimum of sweets reverse the division—67.6 per cent are immune and only 32.4 per cent are carious. This is a difference of 35.2 per cent in favor of a minimal use of sweets.

(d) Similarly, in lines H and I, we find the Excess-of-Sweets: Minimum-of-Sweets ratios in the carious cases (196:23) to be roughly as 10:1, and in the non-carious cases (171:48) to be as 10:3.

TABLES III AND IV. SWEETS AND ITS RELATION TO DENTAL CARIES.

AGES BY YEARS	0-1			1-2			2-3			3-4			4-5			5-6			AVERAGE 0 to 6		
TOTAL MOUTHS BY YEARS	68(6,4%)			212(19,9%)			218(20,4%)			203(19,0%)			203(19,0%)			164(15,4%)			1068(100,0%)		
	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS
D. OR EXCESS OF SWEETS #	2	0	2	42	0	42	99	18	81	121	46	75	121	64	63	117	84	33	367	196	171
%	(3)	0	100	(20)	0	100	(45)	18	82	(60)	38	62	(63)	51	49	(71)	118	282	(64,5)	53,4	46,6
E. ON MINIMUM OF SWEETS #	57	0	57	112	2	110	61	1	60	31	5	26	24	5	19	16	13	3	71	23	48
%	84	0	100	(53)	2	98	(28)	2	98	(15)	14	84	(12)	21	79	(10)	81,3	18,7	(12,4)	32,4	67,6
C. SWEETS IN MODERATION #	9	0	9	58	2	56	58	4	54	31	10	41	30	12	38	31	12	19	132	34	98
%	(13)	0	100	(27)	3	97	(27)	7	93	(25)	20	80	(25)	24	76	(19)	63,2	36,8	(23,1)	25,8	74,2
TOTAL	2	0	2	42	0	42	99	18	81	121	46	75	121	64	63	117	84	33	367	196	171
EXCESSIVE SWEETS	0	0	0	0	0	0	1	1	0	5	5	0	5	5	0	13	13	0	23	23	0
MINIMUM OF SWEETS	0	0	0	2	2	0	60	4	56	26	8	18	19	6	13	3	3	0	48	34	14
MODERATION IN SWEETS	0	0	0	2	0	2	40	13,5	26,5	41	29	12	38	16	22	82	60	22	161	124	37
H - CARIOUS MOUTHS #	0	0	0	4	2	2	23	11	12	46	30	16	24	15	9	71	39	32	196	107	89
%	(0)	0	0	(2)	1	1	(11)	5,5	6	(23)	15	8	(12)	7,5	4,5	(43)	23,8	19,5	(53,4)	29,2	24,2
I - NONCARIOUS MOUTHS #	68	2	66	208	42	166	195	81	114	157	75	82	179	83	96	93	45	48	446	171	275
%	100	3	97	(98)	20	80	(89)	42	58	(77)	37	40	(88)	41	47	(57)	27,4	29,3	(46,6)	38,4	61,6
TOTAL	68	2	66	212	42	170	218	92	126	203	105	98	203	98	105	164	84	80	642	278	364
EXCESSIVE SWEETS	0	0	0	2	2	0	18	18	0	46	46	0	64	64	0	84	84	0	214	214	0
MINIMUM OF SWEETS	57	0	57	110	2	108	60	1	59	26	5	21	19	5	14	3	3	0	275	26	249
MODERATION IN SWEETS	9	0	9	58	2	56	40	4	36	41	17	24	38	12	26	82	60	22	318	124	194
H - CARIOUS MOUTHS #	9	0	9	58	2	56	58	4	54	31	10	21	30	12	18	31	12	19	217	40	177
%	(13)	0	100	(27)	3	97	(27)	7	93	(25)	20	80	(25)	24	76	(19)	63,2	36,8	(23,1)	25,8	74,2
I - NONCARIOUS MOUTHS #	59	2	57	154	40	114	160	14	146	172	95	77	173	86	87	133	72	61	851	238	613
%	(87)	3	84	(73)	19	54	(73)	6	67	(85)	47	38	(85)	42	43	(81)	44	37	(79,6)	22,3	57,4
TOTAL	68	2	66	212	42	170	218	92	126	203	105	98	203	98	105	164	84	80	1068	278	790
EXCESSIVE SWEETS	0	0	0	2	2	0	18	18	0	46	46	0	64	64	0	84	84	0	214	214	0
MINIMUM OF SWEETS	57	0	57	110	2	108	60	1	59	26	5	21	19	5	14	3	3	0	275	26	249
MODERATION IN SWEETS	9	0	9	58	2	56	40	4	36	41	17	24	38	12	26	82	60	22	317	124	193

Fig. 5. Tables III and IV. Ingestion of Sweets and its Relation to Dental Caries. See Graphs III and IV.

(e) The ratios for Caries:Non-caries in this period are indicated below:

	AS SIMPLE RATIOS	IN ACTUAL CASES
For the whole group	8:10	253:317
On excess of sweets	8: 7	196:171
On minimum of sweets	8:17	23: 48
Sweets in moderation	8:23	34: 98

Thus far every advantage is for a minimal or moderate use of sweets.

(f) However, as in the analysis for the benefits of "physiological" oral hygiene, there must be noted here an incidence of caries of 32.4 per cent of all the cases on a minimum of sweets. A minimal use of sweets alone, then, cannot be considered a safe-guard against dental caries.

(2) In the "end-results period," in the analysis for "physiological" oral hygiene, we found decisive contradiction of the tentative deductions only weakly founded upon the 3–6 year period. Precisely the same contradiction is determined in this study of sweets. In the 5–6 year period, just before the children enter their schooling, we find the results indicated below.

(a) The Caries:Non-caries ratios in the 5–6 year group of cases are shown in the appended summary:

	AS SIMPLE RATIOS	IN ACTUAL CASES
For the whole group	20:10	109:55
On excess of sweets	25:10	84:33
On minimum of sweets	43:10	13: 3
Sweets in moderation	6:10	12:19

There is an increased incidence of caries on diets at both extremes of sugar consumption. Indeed, the highest Caries: Immunity ratio found in the whole study (expressed in percentages) is 81:19, which is in this period in the minimum-of-sweets group.

(b) For this age the ratios of cases on excess of sweets (D), to the cases on minimum of sweets (E), to the cases on sweets in moderation (O)—that is, the D:E:O ratios—are shown on page 425.

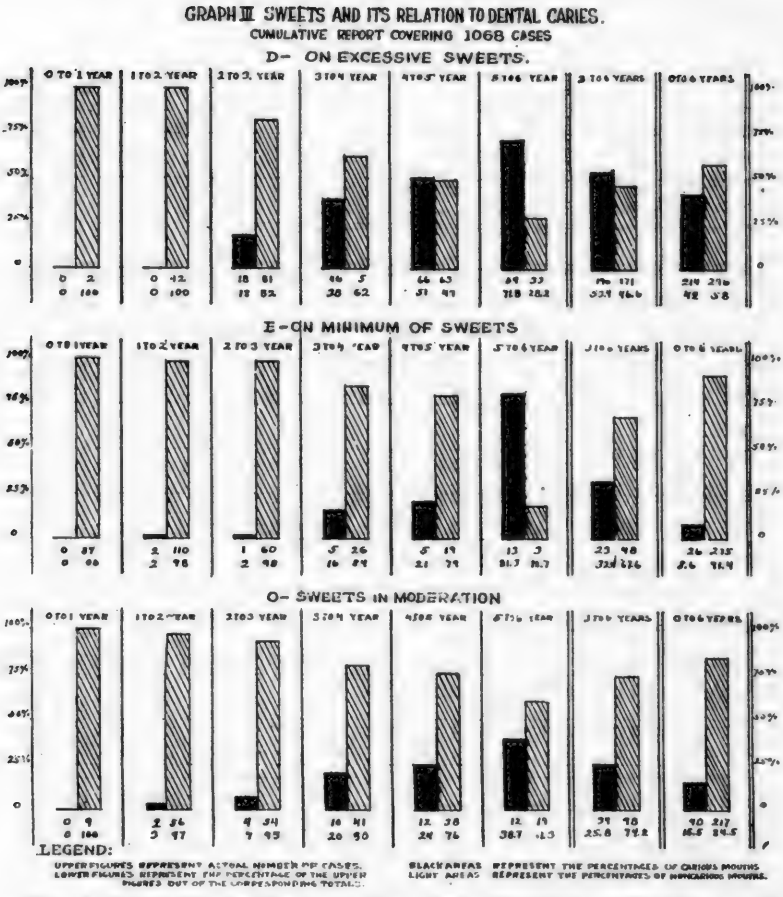
GRAPH III SWEETS AND ITS RELATION TO DENTAL CARIES.
CUMULATIVE REPORT COVERING 1068 CASES
D- ON EXCESSIVE SWEETS.
E-ON MINIMUM OF SWEETS
O- SWEETS IN MODERATION
LEGEND:

FIG. 6. GRAPH III. (SEE TABLES III AND IV.) INGESTION OF SWEETS AND ITS RELATION TO DENTAL CARIES.

Black areas represent the percentages of carious mouths. *Light* areas represent the percentages of non-carious mouths. *D.*—On excess of sweets. *E.*—On a minimum of sweets. *O.*—On sweets in moderation. The first six columns pertain to the successive six years; the seventh and eighth columns, to the 3–6 year and the 0–6 year periods, respectively. The mathematical data are taken from tables III and IV, where they may be studied to greater advantage.

	AS SIMPLE RATIOS	IN ACTUAL CASES
For all cases	20:3: 5	117:16:31
Carious cases	20:3: 3	84:13:12
Non-carious cases	20:2:12	33: 3:19

As before, but a trifling number of cases in the non-carious group are on diets supposedly necessary for tooth preservation. However, the ratio variation between the groups is slight.

(c) In the carious cases (H) the Excess-of-Sweets : Minimum-of-Sweets ratio is as 19:3, and in the non-carious cases (I) this ratio is

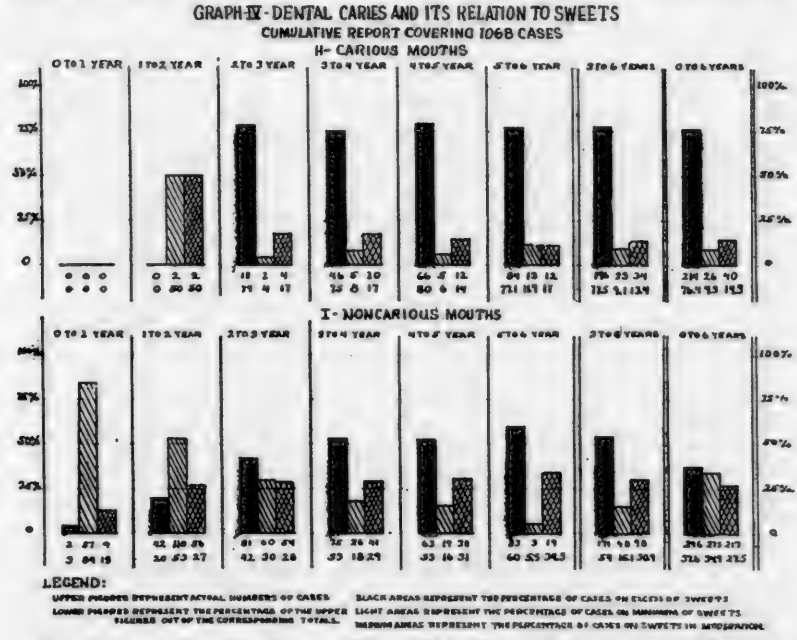
GRAPH IV - DENTAL CARIES AND ITS RELATION TO SWEETS
CUMULATIVE REPORT COVERING 1068 CASES
H- CARIOUS MOUTHS
0 TO 1 YEAR
1 TO 2 YEAR
2 TO 3 YEAR
3 TO 4 YEAR
4 TO 5 YEAR
5 TO 6 YEAR
3 TO 6 YEARS
0 TO 6 YEARS
I - NONCARIOUS MOUTHS
LEGEND:

FIG. 7. GRAPH IV. (SEE TABLES III AND IV.) DENTAL CARIES AND ITS RELATION TO INGESTION OF SWEETS.

Black areas represent the percentages of cases on an excess of sweets. *Light* areas represent the percentages of cases on a minimum of sweets. *Medium* areas represent the percentages of cases on sweets in moderation. *H.*—Carious mouths. *I.*—Non-carious mouths. The first six columns pertain to the successive six years; the seventh and eighth columns, to the 3–6 year and the 0–6 year periods, respectively. The mathematical data are taken from tables III and IV, where they may be studied to greater advantage.

TABLE V "PHYSIOLOGICAL ORAL HYGIENE" AND SWEETS IN COMBINATIONS AS RELATED TO DENTAL CARIES.

AGES BY YEARS			0 - 1			1 - 2			2 - 3			3 - 4			4 - 5			5 - 6			AVERAGE 3 - 6			AVERAGE 0 - 6		
TOTAL MOUTHS BY YEARS			68 (6,4%)			212 (19,9)%			218 (20,4%)			203 (19,0%)			203 (19,0)%			164 (15,4%)			570 (53,5%)			1066 (100,1%)		
			TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS	TOTAL	CARIOUS MOUTHS	NONCARIOUS MOUTHS
- ON A - UNHYGIENIC DIETS	AND ON D — SWEETS IN EXCESS	#	1	0	1	13	0	13	17	3	14	16	5	11	17	11	6	32	23	9	65	39	26	96	42	54
		%	(1,5)	0	100	(6)	0	100	(8)	18	82	(8)	31	69	(8)	65	35	(19,5)	72	28	(11,4)	60	40	(9,0)	(43,8)	56,2
	AND ON E — MINIMUM OF SWEETS	#	2	0	2	3	0	3	3	1	2	2	1	1	0	0	0	2	2	0	4	3	1	12	4	8
		%	(3)	0	100	(1,5)	0	100	1	33	67	1	50	50	(0)	0	0	(1,2)	100	0	(0,7)	75	25	(1,1)	33,3	66,7
	AND ON O — SWEETS IN MODERATION	#	0	0	0	4	1	3	3	1	2	3	1	2	1	1	0	1	0	1	5	2	3	12	4	8
		%	(0)	0	0	(2)	25	75	(1)	33	67	(1,5)	33	67	(0,5)	100	0	(0,6)	0	100	(0,9)	40	60	(1,1)	33,3	66,7
- ON B - HYGIENIC DIETS	AND ON D — EXCESS IN SWEETS	#	0	0	0	0	0	0	8	1	7	6	0	6	7	3	4	10	8	2	23	11	12	31	12	19
		%	(0)	0	0	(0)	0	0	(4)	12	88	(3)	0	100	(3)	43	57	(6,1)	80	20	(4,0)	47,8	52,2	(2,9)	38,7	63
	AND ON E — MINIMUM OF SWEETS	#	1	0	1	7	0	7	22	0	22	11	2	9	10	2	8	7	6	1	28	10	18	58	10	48
		%	(1,5)	0	100	(3)	0	100	(10)	0	100	(5,5)	18	82	(5)	20	80	(4,3)	85,7	14,3	(5,0)	35,7	64,3	(6,5)	17,2	82,8
- ON C - INDIFFERENT DIETS	AND ON O — SWEETS IN MODERATION	#	0	0	0	1	0	1	8	0	8	5	1	4	9	2	7	5	2	1	17	5	12	26	5	21
		%	(0)	0	0	(0,5)	0	100	(4)	0	100	(2,5)	20	80	(4,5)	22	78	(1,8)	66,7	33,3	(3,0)	29,4	70,6	(2,4)	19,2	80,8
	AND ON D — EXCESS OF SWEETS	#	1	0	1	29	0	29	74	14	60	99	41	58	105	52	53	75	61	22	279	146	133	383	160	223
		%	(1,5)	0	100	(14)	0	100	(34)	19	81	(49)	41	59	(51)	50	50	(45,7)	79,3	20,7	(49,0)	52,4	47,6	(35,9)	41,8	58,2
	AND ON E — MINIMUM OF SWEETS	#	34	0	34	201	2	100	36	0	36	15	2	16	14	3	11	7	5	2	39	10	29	231	12	219
		%	(34,5)	0	100	(94,8)	2	98	(16)	0	100	(8,5)	11	89	(7)	21	79	(4,3)	71,4	28,6	(6,8)	25,6	74,4	(21,6)	5,2	94,8
	AND ON O — SWEETS IN MODERATION	#	9	0	9	53	1	52	47	3	44	43	8	35	40	9	31	27	10	17	110	27	83	219	31	188
		%	(13)	0	100	(25)	2	98	(22)	6	94	(21)	19	81	(20)	22	78	(16,5)	37	63	(19,2)	24,6	75,4	(20,5)	14,1	85,9

FIG. 8. TABLE V. "PHYSIOLOGICAL" ORAL HYGIENE AND SWEETS, IN COMBINATION, AS RELATED TO DENTAL CARIES. SEE GRAPH V.

TABLE VI DENTAL CARIES AND IMMUNITY AS RELATED TO COMBINATIONS OF "PHYSIOLOGICAL ORAL HYGIENE" AND SWEETS.

AGES BY YEARS	TOTAL CASES BY YEARS			CARIES & NON-CARIES BY YRS.	CLASSIFICATION GROUPS BY PAIRS								
					A-D	A-E	A-O	B-D	B-E	B-O	C-D	C-E	C-O
0 TO 1	68 6,4%	CARIOUS MOUTHS	#	0	0	0	0	0	0	0	0	0	0
			%	(0)	0	0	0	0	0	0	0	0	0
		NONCARIOUS MOUTHS	#	68	1	2	0	0	1	0	1	54	9
			%	(100)	1,5	3	0	0	1,5	0	1,5	79,5	13
1 TO 2	212 19,9%	CARIOUS MOUTHS	#	4	0	0	1	0	0	0	0	2	1
			%	(2.0)	0	0	25	0	0	0	0	50	25
		NONCARIOUS MOUTHS	#	208	13	3	3	0	7	1	29	100	52
			%	(98)	6.2	1,5	1,5	0	3,3	0,5	14	48	25
2 TO 3	218 20,4%	CARIOUS MOUTHS	#	23	3	1	1	1	0	0	14	0	3
			%	(11)	13	4,3	4,3	4,4	0	0	61	0	13
		NONCARIOUS MOUTHS	#	195	14	2	2	7	22	8	60	36	44
			%	(89)	7	1	1	4	11	4	31	18	23
3 TO 4	203 19,0%	CARIOUS MOUTHS	#	61	5	1	1	0	2	1	41	2	8
			%	(30)	8	2	2	0	3	2	67	3	13
		NONCARIOUS MOUTHS	#	142	11	1	2	6	9	4	58	16	35
			%	(70)	7,8	0,7	1,4	4,2	6,3	2,8	40,8	11,2	24,8
4 TO 5	203 19,0%	CARIOUS MOUTHS	#	83	11	0	1	3	2	2	52	3	9
			%	(41)	13,2	0	1,2	3,6	2,4	2,4	62,6	3,6	11
		NONCARIOUS MOUTHS	#	120	6	0	0	4	8	7	53	11	31
			%	(59)	5	0	0	3	7	6	44	9	26
5 TO 6	164 15,4%	CARIOUS MOUTHS	#	109	23	2	0	8	6	2	53	5	10
			%	(67)	21,2	1,8	0	7,3	5,5	1,8	48,6	4,6	9,2
		NONCARIOUS MOUTHS	#	55	9	0	1	2	1	1	22	2	17
			%	33	16,4	0	1,8	3,6	1,8	1,8	40	3,6	30,9
AVERAGE 3 TO 6	570 53,5%	CARIOUS MOUTHS	#	253	39	2	2	11	10	5	148	27	10
			%	(44,4)	15,1	1,2	0,8	4,3	4	2	57,7	4	10,7
		NONCARIOUS MOUTHS	#	317	26	1	3	12	18	12	133	29	83
			%	(55,6)	8.2	0.3	1	3.8	5.7	3.8	42	9.2	26.2
AVERAGE 0 TO 6	1068 100,1%	CARIOUS MOUTHS	#	280	42	4	4	12	10	5	160	12	31
			%	(26,2)	15	1,4	1,4	4,3	3,6	1,8	57,2	4,3	11
		NONCARIOUS MOUTHS	#	788	54	8	8	19	48	21	227	219	188
			%	(73,8)	6,9	1	1	2,4	6,1	2,7	28,1	27,8	23.8

FIG. 10. TABLE VI. DENTAL CARIES AS RELATED TO "PHYSIOLOGICAL" ORAL HYGIENE AND SWEETS, IN COMBINATION. SEE GRAPH VI.

as 33:3 (84:13 cases, and 33:3 cases, respectively). This represents almost the reversal of the findings one should expect, if a minimal use of sugar prevented caries and a maximal use of sugar induced it.

(d) Analysis of the critical figures relating to the use of sweets in relation to dental caries shows them to be much the same as those found for "physiological" oral hygiene. The ratios in all citations are nearly enough the same, in the children studied, again to indicate

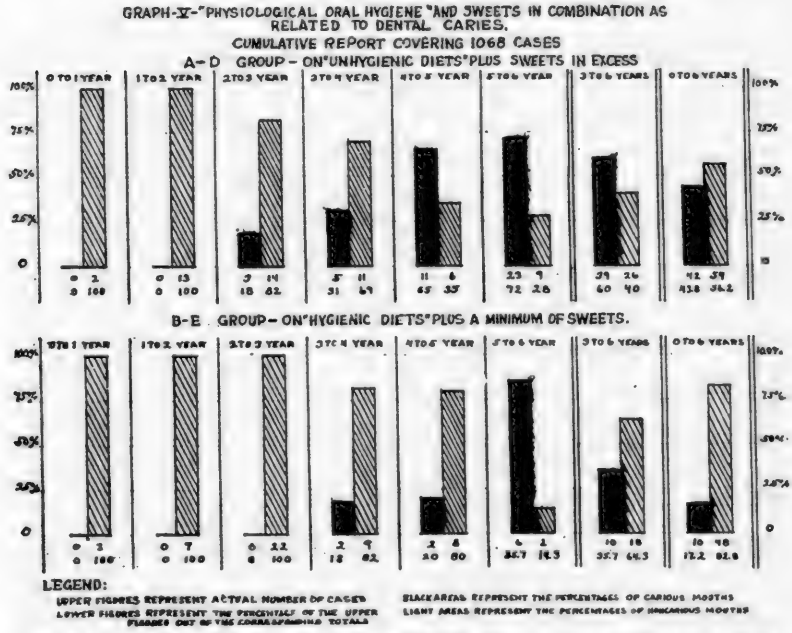

FIG. 9. GRAPH V. (SEE TABLE V.) "PHYSIOLOGICAL" ORAL HYGIENE AND SWEET IN COMBINATION, AS RELATED TO DENTAL CARIES.

Black areas represent the percentages of carious mouths. *Light* areas represent the percentages of non-carious mouths. *A–D group.*—On "unhygienic" diets plus sweets in excess. *B–E group.*—On "hygienic" diets plus a minimum of sweets. The first six columns pertain to the successive six years; the seventh and eighth columns, to the 3–6 year and the 0–6 year periods, respectively. The mathematical data are taken from table V, where they may be studied to greater advantage.

a fairly constant incidence of dental caries, regardless of indulgence in sweets or abstinence therefrom.

Thus, critical examination of the crucial year of this study fails to reveal any evidence supporting the doctrine that free sugars tend to initiate dental caries, and that a minimal indulgence lends protection.

v. Sweets plus "Physiological" Oral Hygiene and Dental Caries

The possible combinations of classes in pairs, between the two groups, are nine in number. Each is indicated by two letters, and is arranged in tables V and VI. There are two combinations which

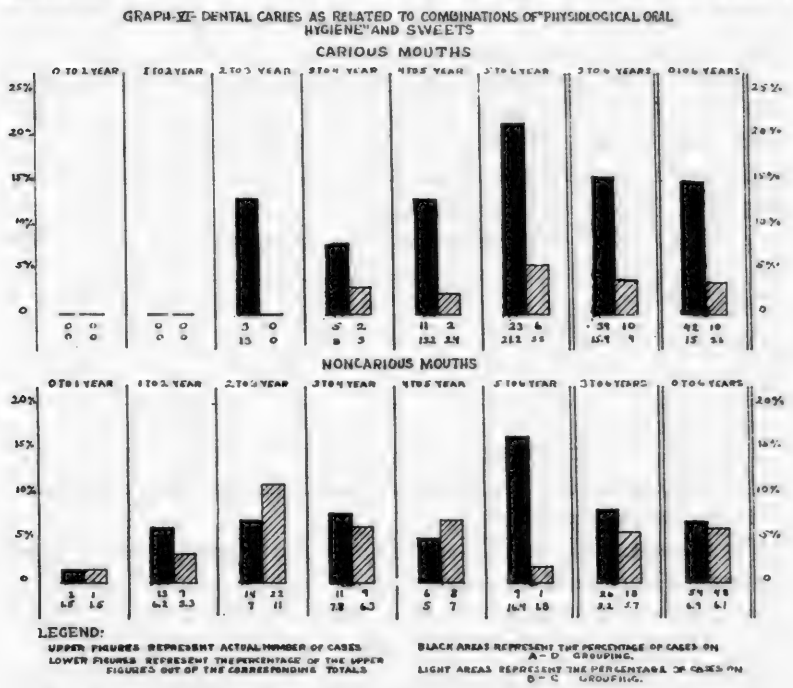
GRAPH-VI- DENTAL CARIES AS RELATED TO COMBINATIONS OF "PHYSIOLOGICAL ORAL HYGIENE" AND SWEETS
CARIOUS MOUTHS
NONCARIOUS MOUTHS
LEGEND:
UPPER FIGURES REPRESENT ACTUAL NUMBER OF CASES
LOWER FIGURES REPRESENT THE PERCENTAGE OF THE UPPER FIGURES OUT OF THE CORRESPONDING TOTALS
BLACK AREAS REPRESENT THE PERCENTAGE OF CASES ON A-D GROUPING.
LIGHT AREAS REPRESENT THE PERCENTAGE OF CASES ON B-E GROUPING.

FIG. 11. GRAPH VI. (SEE TABLE VI.) DENTAL CARIES AS RELATED TO "PHYSIOLOGICAL" ORAL HYGIENE AND SWEETS, IN COMBINATION.

Black areas represent the percentages of cases in the A–D group. *Light* areas represent the percentages of cases in the B–E group. *Upper half.*—Carious mouths. *Lower half.*—Non-carious mouths. The first six columns pertain to the successive six years; the seventh and eighth columns, to the 3–6 year and the 0–6 year periods, respectively. The mathematical data are taken from table VI, where they may be studied to greater advantage.

should represent the extremes of the bad and the good groupings, namely, A–D and B–E. These are the unhygienic-plus-excess-sugar grouping and the hygienic-plus-minimum-of-sugar grouping, respectively.

Only one citation need be made from the 3–6 year column: that is to note that, with the combined use of the two dietaries, each recommended as prophylactic against caries, there is still to be found an appreciable incidence of decay—of the thirty-one mouths thus safeguarded, 35.7 per cent of them present the disease obviously.

Turning attention now to the "end-results period"—five to six years of age—the following may be noted.

(a) For all the cases seen (164), again disregarding their groupings, the Caries:Immunity ratio is 109:55, or approximately as 20:10. This ratio, in the A–D grouping, is as 26:10; in the B–E grouping, it is more than doubled and is as 60:10. (In actual cases represented they are 23:9, and 6:1, respectively.)

(b) The A–D:B–E ratios in the 5–6 year period are shown below:

	AS SIMPLE RATIOS	IN ACTUAL CASES
For all cases	46:10	32:7
Carious cases	38:10	23:6
Non-carious cases	90:10	9:1

This means that the supposedly bad diet combination, *i.e.*, the A–D grouping, is used by a larger fraction in the non-carious group of children than in the carious.

Thus it is seen that our data give no greater support to advantages that might be claimed for "physiological" oral hygiene and a minimal use of sweets, in combination, than when considered singly. Even when their proposed advantages should be mutually strengthening, if used in combination in actual practice, the desired results again are not borne out by the findings herein recorded.

VIII. RÉSUMÉ OF THE DISCUSSION

(1) The mass incidence of dental caries begins at the third year, so the last three years are grouped for study as the "age of the mass incidence of caries." The most informative facts should be found,

then, in the cases from three to six years of age. Analysis of this period gives some support to the two theories. It presents a decreased incidence of carious mouths among the children on diets that fit the description of Wallace for "physiological" oral hygiene (21.3 per cent less than on unhygienic diets). There is also a decreased incidence among the children using a minimal amount of sweets (21.0 per cent less than among those using excess of sweets).

(2) However, the validity of the findings is somewhat decreased by the presence of considerable caries among the children on the advocated diets; 38.2 per cent on "physiological" oral-hygiene diets showed caries; 32.4 per cent of those on a minimum of sweets; and 35.7 per cent of those on these diets in combination. Application of the theories did not completely protect against the disease.

(3) The incidence of caries rises rapidly in the 3–6 year period—from 30 per cent to 67 per cent. Thus, findings in the early part may considerably alter results properly derived from the "end-results period," as the last age period of the study has been termed.

To this 5–6 year period the following facts apply:

(a) The Caries:Non-caries ratios are highest in the groups of cases on supposedly protective diets, both singly and in combination, as follows:

	CARIES:NON-CARIES
For all cases	20:10
On unhygienic diets	25:10
On hygienic diets	40:10
On excess of sweets	25:10
On minimum of sweets	43:10
Most unfavorable group (A–D)	26:10
Most favorable group (B–E)	60:10

(b) In each instance the ratio of the condemned diet to the advocated diet is found to be the highest in the non-carious groups of cases, as follows:

	UNHYGIENIC TO HYGIENIC	EXCESS OF SWEETS TO MINIMUM SWEETS	A–D GROUP TO B–E GROUP
All cases	5:3	23:3	46:10
Carious cases	5:3	19:3	38:10
Non-carious cases	5:2	33:3	90:10

(4) The variations of this "end-results period" serve to guard against equivocal interpretations that might have arisen had the meagre differences chanced to be on the side advantageous to the theories. But, for the method employed, the differences are so small that they may be all but disregarded. The disproportions are not significant. The study, then, seems to indicate that there is a fairly constant incidence of caries and of immunity in children by the time they reach their school years, regardless of "physiological" oral hygiene of the diets (as defined) and of indulgence in sweets.

IX. SUMMARY AND CONCLUSIONS

Most of the dietetic theories of dental caries speculate too freely upon the anticipated eradication of the disease. Their proponents have not determined whether application of the theories actually brings about the desired results.

The statistical method for a large number of cases, if dealt with without bias, should be of distinct value in such studies. A thesis should gain considerable weight, if controlled study bears it out. Contrariwise, negative findings materially reduce the force of a hypothesis.

The present study included examinations of 1,068 children of preschool age. It is an inquiry into the objective results of application of two prominent theories on the causation and prevention of dental caries. They are the "physiological-oral-hygiene" theory of Wallace and the well-known dictum that sweets decay teeth. Each theory is carefully defined by quotations from proponents.

Methods of study, and the classification and interpretation of data, are described in detail. This is a study of caries obvious to direct inspection without instrumentation.

Each theory is critically considered upon the basis of collected statistical data. The effect of application of the two theories in

combination, which should lend them mutual support, is similarly studied.

The analyses of the cases collected are detailed at length in section VII. Section VIII is a résumé of that section.

Since the theories studied are not borne out at all by the clinical-statistical method employed, their validity must be held in some doubt until otherwise fortified with equally reliable methods or data supporting them. This is not a contention that the theories have been disproved or completely invalidated. It indicates only that their validity has been challenged. Until we have results of further careful research upon them, we must be cautious not to be converted by the mere pictorial impressiveness of the theses. The every-day findings of wholesale decay of teeth in a people living on our artificial dietaries, or who lavishly indulge in candies, jams and sugars, should not alone, or without carefully controlled comparison series, persuade us prematurely to conclusions on this important question.

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BLOOD CHEMISTRY AS A DIAGNOSTIC AID IN FOCAL INFECTION[1]

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I. INTRODUCTION

I am very grateful for the opportunity to meet you tonight, and to address you upon the subject of focal infection—a subject of vital importance to both medical and dental professions. This subject, while not new, is only beginning to come into its own. As has often happened in the past, the introduction of a new theory has brought about a division of the professions into several groups based on some of the apparently contradictory phenomena involved. It is my hope that blood chemistry, when properly utilized, will help to clear away some of these supposed paradoxes, and enable all of us to unite on a common basis and a more complete understanding.

At present our professions appear to be divided into three groups: the conservatives, who fail to accept the focal-infection theory either in whole or in part; the radicals, who jump to conclusions and in their enthusiasm are apt to overdo the matter of eradication of supposed foci; and, finally, the group (altogether too small) who realize that both conservatism and radicalism have their places.

[1] Read at the ninth annual meeting of the American Academy of Periodontology, Hotel Gibson, Cincinnati, December 6, 1922. See the related paper by McCall, and the ensuing discussion: *Journal of Dental Research*, 1922, iv; proceedings section, p. lxxi (this issue).

The chief object of this paper is to bring before you the fact that we now have a very definite means of diagnosing focal infection through blood chemistry, and thus settling many of the perplexing problems of chronic systemic disease. This does not mean that the chemical findings determine whether the focus is present in teeth, tonsils, or sinuses; the findings indicate only the presence or absence of foci *in the body*, and thus may justify what is frequently a prolonged and difficult search. I base this statement on my findings during the past seven years, on my own patients and on those referred to me for diagnosis. Very close coöperation of certain types of medical and dental specialists is necessary to localize definitely the focus or foci of infection that chemical study has proved to be present. The groups of specialists most generally called upon, to localize and eradicate such infection, include the laryngologist, the dental roentgenologist, the oral surgeon, and the internist; the general surgeon is sometimes needed as well.

In my opinion a focus of infection may best be defined as a circumscribed mass of nuclear degeneration, which may or may not be encysted. This may mean a chronic accumulation of pus, or it may mean simply a region exhibiting an abnormal degeneration of cellular elements. This mass may be enclosed in any of the cavities or tissues of the body. The tissues most commonly involved are the tonsils, including all lymphoid tissue in Waldeyer's ring; the teeth and periodontal tissues; the nasal accessory sinuses; the middle ear; the mastoid and lateral sinuses; in fact, all the cavities communicating with the mouth, nose, and naso-pharynx. I have arranged this classification in an order indicating the usual relative degrees of importance, namely, tonsils, teeth, and sinuses. The secondary results of focal infection in the body (and these constitute the patient's chief source of complaint) are indeed legion, but I will not burden you with allusions to them at this time.

The primary focus of infection must be regarded not only as the place of entrance for the bacteria, but also the place where the organisms acquire the peculiar properties necessary for infection. The location of such foci frequently offers one of the most difficult problems in medicine.

There are two distinct methods of approaching the study of focal infection, namely: bacteriological and chemical. It is not my intention to discuss the bacteriological phase, with its many interesting channels, the most noted chapter of which is Rosenow's work on the selective affinity of given strains of organisms for certain tissues. The organisms, or the toxins produced by these organisms, from one or more foci, give rise to the various clinical pictures so frequently seen in a certain class of patient. The bacteriological studies of Rosenow have proven conclusively that primary infection can exist about a tooth, and in the periodontal tissues, and is capable of giving rise to various secondary infections throughout the body. The relationship he has demonstrated between primary and secondary foci shows the importance of complete eradication of existing primary foci of infection.

II. ON THE PRODUCTION OF URIC ACID IN FOCI OF INFECTION: CAUSES OF HYPER-URIC-ACIDEMIA

In our chemical studies we have found a method of determining the existence of a focus or foci of infection in the body, thus providing a means of applying the focal-infection theory to the individual case. It is my desire to express myself as simply as possible, but it will be necessary to use some chemical terms that are more or less familiar to all of you. It is necessary, first, clearly to understand that the blood is a tissue in intimate contact with all other tissues, in order that we may appreciate its value in indicating the presence of infection in any portion of the body. The normal "chemical" constituents of the blood may be divided into the *organic salts*, of proteolytic origin; *inorganic salts*, ingested as such; *carbohydrates*, from starches and sugar; *lipoids*, from cholesterols and fats. At this time we will consider only the constituents of proteolytic origin, not that the others are unimportant but because they do not enter into the present discussion. The studies of these other factors are, however, of inestimable value as indicating the extent of changes, if any, in the general metabolism of the patient. These other substances should, therefore, always be included in any chemical studies of the blood, to ascertain whether their concentration is outside of normal limits.

We have stated that focal infection is essentially a process leading to nuclear degeneration. This, in a word, means the production of nucleic acid. To elaborate somewhat: an infective process in a circumscribed tissue region results in a degeneration and possibly in a destruction of the cellular elements of the part, of the cellular elements of the blood, and of the bacteria themselves. All cells consist of protoplasm, a part of which is differentiated from the rest of the cell and constitutes the nucleus. One of the characteristic chemical substances found in cell nuclei is nucleic acid. The degeneration of cells and their nuclei gives rise to the liberation of "split" proteins and nucleic acid. The amount of nucleic acid produced depends on the number of existing foci, on the virulence of the infecting agent, and on the resistance of the host. We start, therefore, with nucleic acid and study its fate in the organism. This brings us to a consideration of the group of substances of which nucleic acid or its immediate forerunner, nucleoprotein, is a member, namely, the protein group. The metabolic end-products of this group are usually stated to be creatinin, urea, and uric acid; and it is from nucleoprotein or nucleic acid that uric acid is derived.

It will now be necessary to consider, for a moment, the chief avenue of elimination, namely the kidneys. We find that excretion, by the kidneys, of these various metabolic end-products occurs in the following order: first, creatinin, the most diffusible; then urea, intermediate; and uric acid, the least diffusible. For this reason these end-products have long been used as indices of renal permeability or function only. We find, however, that an exceedingly large number of individuals have an increased content of uric acid, with a normal or only moderately increased proportion of urea, in the blood. As further proof that such cases of hyper-uric-acidemia are not of renal origin, we find a normal urine with no fixation of the specific gravity, and no nocturia. The self-evident conclusion, from a series of such cases, is that the increased content of uric acid in the blood is not due to kidney damage, but rather to over-production of this end-product of nuclear degeneration.

Having thus briefly eliminated the kidney as the cause of hyper-uric-acidemia, we can therefore refrain from further mention of urea and creatinin. This brings us, then, to the point of our discussion,

namely, uric acid. We will consider briefly its two sources of origin, exogenous and endogenous. The exogenous sources are the foods rich in nucleic acid and commonly known as the purin group of foods. In this group are meats, legumes, and the edible glands of animals. It is impossible, however, for an individual to consume enough of these foods to produce a persistently high content of uric acid in the blood from this source alone. In other words, when renal function is normal, as indicated by the blood findings for urea and creatinin, the ingestion of food rich in purin bases merely produces a temporary increase in the uric acid in the blood, this uric acid being passed off through the kidneys in a short time. We can, therefore, further eliminate diet as a causative factor for a persistently high content of uric acid in the blood.

The endogenous uric acid arises from katabolism of body cells, and may be normal or abnormal. Such breaking down of cellular elements, when abnormal in amount, is found in a number of conditions, other than focal infections, such as primary anemias, leukemias, severe cachexias following large doses of x-ray or radium. It follows, then, that the conditions which may cause a hyper-uric-acidemia not or focal-infection origin can be eliminated with comparative ease.

III. THE PROPORTION OF URIC ACID IN THE BLOOD AS A DIAGNOSTIC INDEX OF THE EXISTENCE OF FOCAL INFECTION (AFTER EXCLUSION OF OTHER CAUSES OF HYPER-URIC-ACIDEMIA)

In cases in which examination makes it possible to eliminate the foregoing factors, it may be stated that a high content of uric acid in the blood is due to nuclear degeneration, which may be due to focal infection. In fact, if the aforementioned endogenous sources of uric acid are eliminated, such a high blood content must be due to focal infection. This finding makes the data of blood chemistry of inestimable value in the diagnosis of focal infection. We find, for instance, many evidences of infection in the mouth in regions where degeneration is not sufficiently pronounced to be macroscopic, and which have not reached the stage of definite abscess formations with well defined walled-off masses of pus. Such regions, however, invariably present microscopic evidence of inflammatory changes. The periodontal bone

may exhibit rarefaction or condensation, or may show only a change in the arrangement of the cancellous spaces. The variation of such regions, in density or structure, from the normal bone surrounding them may be so slight as readily to escape detection. To my mind, these regions are even more dangerous than those marked off by a layer of dense bone or pyogenic membrane, as they allow toxins to be absorbed freely into the circulation. These regions of diffuse rarefying osteitis and diffuse condensing osteitis, or those that exhibit alterations in the cancellation of the bone, produce virtually the same leukocytic infiltration, the same degeneration of tissue and blood elements, the same liberation of "split" proteins, as does a well defined abscess.

Too often has disappointment for both practitioner and patient followed a diagnosis that was incomplete rather than incorrect. Not only in the mouth, but also in other parts of the body, a definite diagnosis is hard to make. It is safe to say that the major part of the present scepticism regarding the reality of focal infection rests on this point. It is in this respect that chemical studies play so important a rôle. Such blood studies are also extremely valuable as a check on the thoroughness of eradication of the foci found, for, as long as any focus is present, the uric acid findings will not return to a normal level. This search for foci, let me repeat, is frequently most difficult and may require the most intimate form of team work of all concerned. Practice, however, based upon this method, has been most happy in its results. Patients invariably show such a measure of improvement as is to be expected from their history, and in accordance with the regenerative power of the tissues at the sites of the secondary infections.

IV. SUMMARY OF CONCLUSIONS

(1) Clinicians have at their disposal well established methods for the estimation of uric acid in blood.

(2) A persistently high uric-acid value for the blood is indicative of nuclear degeneration, which in turn may mean focal infection.

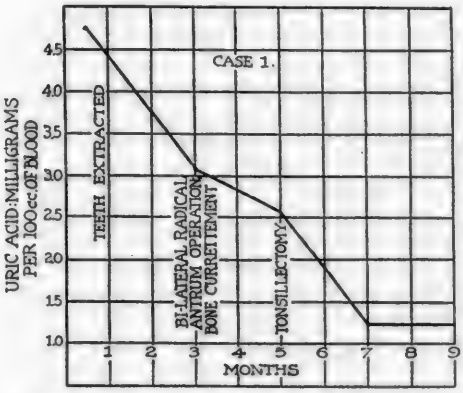
(3) Other factors productive of a high uric-acid value for the blood, aside from nuclear degeneration, are comparatively easy to determine, namely, leukemia, primary anemias, cachexias from whatever cause, and massive doses of x-ray or radium.

(4) Elimination of all foci of infection is followed, invariably, by a return to normal of the uric-acid value for the blood.

(5) Failure to eliminate all foci prevents return to a normal uric-acid value for the blood. The amounts of this substance, therefore, furnish a reliable index for the complete elimination of foci of infection.

V. CASE HISTORIES, WITH CHEMICAL DATA

The following brief case histories, and the accompanying graphs and collateral analytic data, give typical examples of the blood findings and the clinical parallels, selected from a series of 827 cases.



CASE 1. THE AUTHOR. PRONOUNCED ASTHENIA

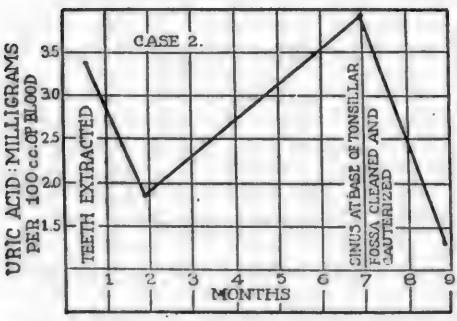
Case 1: Collateral analytic data[2]

	Milligrams per 100 cc. of blood First	Last
Urea	45	30
Urea N	21	14
Total non-protein N	42	28
Creatinin	0.4	0.2
Salt	500	500
Sugar	155	135
Cholesterol	210	175
Fatty acids	800	725
Total fats	1010	900
CO_2 combining power	47.2	49.0
Diastatic activity	31	21
Wassermann test	neg.	neg.
Plasma volume (%)	52	50
Corpuscle volume (%)	48	50

Case 1. The author. Pronounced asthenia. My own case was responsible for my initial interest in the causation of high blood content of uric acid. The physical symptoms could be summed up as indicating a pronounced asthenia. Extraction of several badly infected teeth brought some improvement and a decrease in the blood content of uric acid. A bi-lateral radical antrum operation brought about a still further improvement. The final return to the normal uric-acid level, and to the normal physical condition, was not secured until after tonsillectomy.

[2] In these nine summaries, "first" signifies the initial finding; "last," the finding at the concluding examination. "N" signifies nitrogen. For references to the method of determining uric acid, used by the author, see Myers and Fine: *Journal of Biological Chemistry*, 1915 (xx); and Folin and Wu: *Ibid.*, 1919 (xxxviii).

Case 2. Mrs. F.: age 42. Chronic arthritis. The complaint was pain in the knees, shoulders and elbows, making it difficult for her to help herself; easy fatigue or asthenia. The tonsils had been removed and the throat appeared to be in good condition. Radiograms showed six infected teeth; their removal, with currettement, was followed by great improvement. This patient returned about one year later in greater discomfort than ever, with a higher uric-acid content in the blood than primarily. Investigation showed that the teeth and periodontal tissues were clear. Quite accidentally, in investigating the tonsillar fossa (which appeared clear), a sinus about three-fourths of an inch long was found, filled with purulent material (from which streptococcus viridans was isolated). This was cleaned out, cauterized and kept clean. Recovery quickly followed.



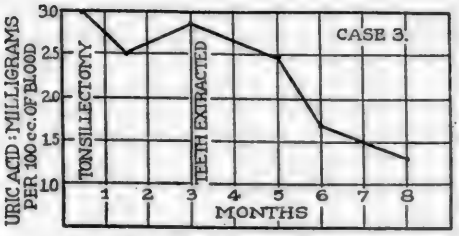
CASE 2. MRS. F.: AGE 42. CHRONIC ARTHRITIS

Case 2: Collateral analytic data

	Milligrams per 100 cc. of blood First	Last
Urea	60	45
Urea N	28	21
Total non-protein N	56	42
Creatinin	0.6	0.4
Salt	510	500
Sugar	180	145
Cholesterol	260	220
Fatty acids	980	840
Total fats	1240	1060
CO_2 combining power	47.2	48.4
Diastatic activity	16	25
Wassermann test	neg.	neg.
Plasma volume (%)	58	54
Corpuscle volume (%)	42	46

Case 3. Miss B.: age 34; school teacher. Arthritis. She had been under the care of an orthopedist for two years and was getting progressively worse. Had pain in and some swelling of the ankles, knees and shoulders, so that she had not been able to teach for eighteen months. Some badly infected tonsils were removed, with very slight improvement. She had four definitely infected teeth but did not wish to lose them, as she had just had two fine removable bridges completed at considerable expense. She finally consented to extraction and currettement. Inside of two months there was complete recovery from all symptoms.

Case 4. Mr. H.: age 46. Typical gout. He had some kidney and cardiac complications. He regarded extraction of teeth as a fad, but consented to have three teeth taken out because of local discomfort. There

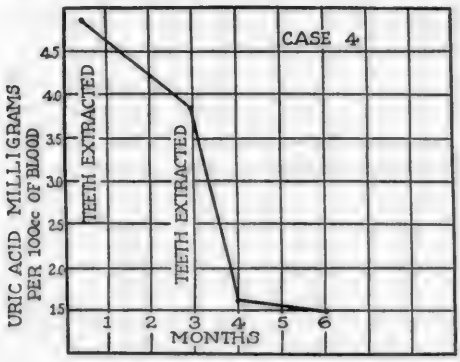


CASE 3. MISS B.: AGE 34. ARTHRITIS

Case 3: Collateral analytic data

	Milligrams per 100 cc. of blood First	Last
Urea	45	30
Urea N	21	14
Total non-protein N	42	28
Creatinin	0.45	0.3
Salt	460	480
Sugar	160	150
Cholesterol	210	190
Fatty acids	840	710
Total fats	1050	1000
CO_2 combining power	47.6	49.0
Diastatic activity.	20	24
Wassermann test.	neg.	neg.
Plasma volume (%)	50	50
Corpuscle volume (%)	50	50

was very slight improvement. After six months he was willing to proceed further and his oral condition was cleaned up. Recovery from the gouty symptoms was uneventful. His renal and cardiac conditions also improved somewhat, although they are still present.



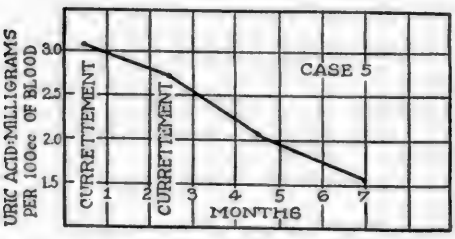
CASE 4. MR. H.: AGE 46. TYPICAL GOUT

Case 4: Collateral analytic data

	Milligrams per 100 cc. of blood First	Last
Urea	75	45
Urea N	35	21
Total non-protein N	70	42
Creatinin	0.6	0.3
Salt	540	490
Sugar	200	160
Cholesterol	230	175
Fatty acids	640	640
Total fats	870	815
CO_2 combining power	47.2	48.0
Diastatic activity.	10	18
Wassermann test.	neg.	neg.
Plasma volume (%)	60	52
Corpuscle volume (%)	40	48

Case 5. Mrs. L. W.: age 48. Diabetes mellitus and arthritis. The patient was in a general subnormal condition, with a very low carbohydrate tolerance. The tonsils had been removed some time previously. All her teeth had been removed. Radiograms, however, revealed several regions of infection in the bone and about two small roots. After currettement, another radiogram was taken and further currettement was done. There

was a slight improvement in her carbohydrate tolerance, although rigid diet is still necessary. The patient also completely recovered from the arthritic condition. Whether this was due to the improvement in her diabetic condition, or to the clearing up of focal infection, I am not prepared to say.

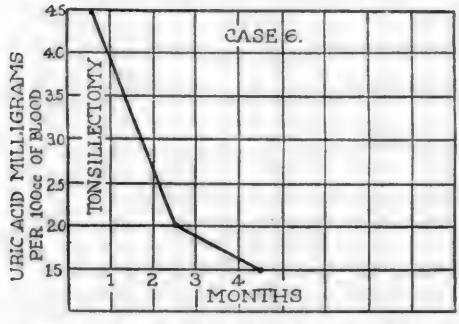


CASE 5. MRS. L. W.: AGE 48. DIABETES MELLITUS AND ARTHRITIS

Case 5: Collateral analytic data

	Milligrams per 100 cc. of blood First	Last
Urea	60	45
Urea N	28	21
Total non-protein N	56	42
Creatinin	0.3	0.22
Salt	480	485
Sugar	420	200
Cholesterol	240	200
Fatty acids	910	880
Total fats	1150	1080
CO_2 combining power	46.8	47.8
Diastatic activity.	12	18
Wassermann test.	neg.	neg.
Plasma volume (%)	56	50
Corpuscle volume (%)	44	50

Case 6. Miss B.: age 12. Chorea of two years' duration. The tonsils were found to be badly infected and removed. Recovery was uneventful.



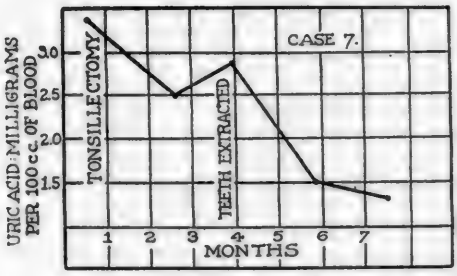
CASE 6. MISS B.: AGE 12. CHOREA OF TWO YEARS' DURATION

Case 6: Collateral analytic data

	Milligrams per 100 cc. of blood First	Last
Urea	30	30
Urea N	14	14
Total non-protein N	28	28
Creatinin	0.15	0.2
Salt	430	450
Sugar	140	135
Cholesterol	175	200
Fatty acids	625	600
Total fats	800	800
CO_2 combining power	48.0	48.8
Diastatic activity.	22	24
Wassermann test.	neg.	neg.
Plasma volume (%)	50	50
Corpuscle volume (%)	50	50

Case 7. Mr. J.: age 40. Asthma of six years' duration; not seasonal. This patient was not sensitive to any of the food proteins. Blood analysis showed focal infection. The tonsils were manifestly bad. Tonsillectomy

was followed by slight improvement. This patient would not immediately permit removal of the five teeth indicated on the radiograms. Later, there was recurrence of the symptoms. As the blood data still showed the presence of focal infection, he submitted himself to the oral surgeon. Recovery, to date.

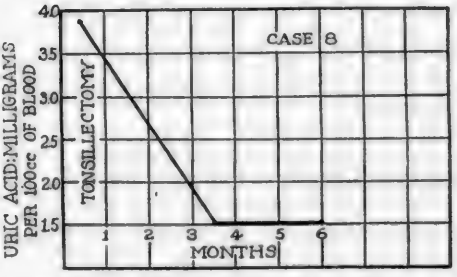


CASE 7. MR. J.: AGE 40. ASTHMA OF SIX YEARS' DURATION; NOT SEASONAL

Case 7: Collateral analytic data

Milligrams per 100 cc. of blood	*First*	*Last*
Urea	60	30
Urea N	28	14
Total non-protein N	56	28
Creatinin	0.42	0.31
Salt	490	485
Sugar	160	152
Cholesterol	180	200
Fatty acids	740	710
Total fats	920	910
CO_2 combining power	49.0	49.4
Diastatic activity	20	24
Wassermann test	neg.	neg.
Plasma volume (%)	54	50
Corpuscle volume (%)	46	50

Case 8. Miss E.: age 16. A 3-plus enlargement of the thyroid gland, bi-lateral; also some exophthalmos. There was no tremor but a tachycardia, and disturbance of menstruation, with a plus-12 basal metabolism. The tonsils were infected. Tonsillectomy was followed by slow recovery for six months, when she made complete recovery.

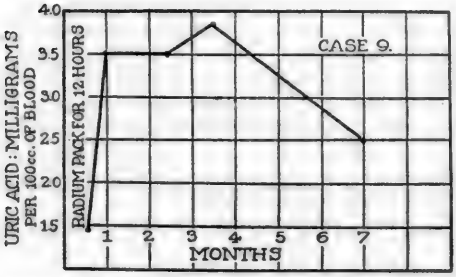


CASE 8. MISS E.: AGE 16. A 3-PLUS ENLARGEMENT OF THE THYROID GLAND, BILATERAL; ALSO SOME EXOPHTHALMOS

Case 8: Collateral analytic data

Milligrams per 100 cc. of blood	*First*	*Last*
Urea	30	30
Urea N	14	14
Total non-protein N	28	28
Creatinin	0.2	0.2
Salt	480	470
Sugar	135	140
Cholesterol	190	200
Fatty acids	820	700
Total fats	1010	900
CO_2 combining power	49.2	49.0
Diastatic activity	20	22
Wassermann test	neg.	neg.
Plasma volume (%)	48	50
Corpuscle volume (%)	52	50

Case 9. Mrs. Y.: age 58. The patient had a radium pack for a fibroid tumor. The rise in blood content of uric acid was followed by a partial return to normal. This case is shown simply to illustrate the effects of radium and x-rays on metabolism.

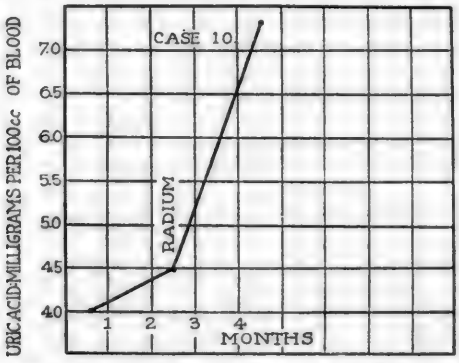


CASE 9. MRS. Y.: AGE 58. THE PATIENT HAD A RADIUM PACK FOR A FIBROID TUMOR

Case 9: Collateral analytic data

	Milligrams per 100 cc. of blood	
	First	*Last*
Urea	45	96
Urea N	21.0	44.8
Total non-protein N	42.0	89.6
Creatinin	0.42	0.7
Salt	490	500
Sugar	160	170
Cholesterol	190	200
Fatty acids	860	740
Total fats	1050	960
CO_2 combining power	48.8	46.8
Diastatic activity.	24	18
Wassermann test	neg.	neg.
Plasma volume (%)	52	58
Corpuscle volume (%)	48	42

Case 10. Mr. L.: age 32. Acute lymphatic leukemia. Uric acid content in the blood was high when first determined, and increased steadily. It rose still more rapidly after radium treatment. The case illustrates



CASE 10. MR. L.: AGE 32. ACUTE LYMPHATIC LEUKEMIA; TREATMENT WITH RADIUM

the effect of this condition on the amount of uric acid in the blood. This case, and case 9, illustrate conditions that have to be eliminated in arriving at a diagnosis of focal infection from the data for uric acid in the blood.

482 Franklin Street

SCIENTIFIC PROCEEDINGS

OF

DENTAL AND STOMATOLOGICAL SOCIETIES

A LIST OF THE NAMES OF THE ORGANIZATIONS THAT HAVE VOTED TO MAKE THE JOURNAL OF DENTAL RESEARCH THE MEDIUM FOR THE OFFICIAL PUBLICATION OF THEIR SCIENTIFIC PROCEEDINGS

(December, 1922)

ASSOCIATION OF THE ALLIED DENTAL SOCIETIES, INC.

AMERICAN ACADEMY OF DENTAL SCIENCE

MASSACHUSETTS DENTAL SOCIETY

HARVARD ODONTOLOGICAL SOCIETY

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FIRST DISTRICT DENTAL SOCIETY OF THE STATE OF NEW YORK

METROPOLITAN DISTRICT OF THE MASSACHUSETTS DENTAL SOCIETY

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In the case of the last named organization, only the dental and stomatological parts of its proceedings will be published in this JOURNAL.

A standing general invitation is extended to dental and stomatological societies, everywhere, to add their names to the above register.

SCIENTIFIC PROCEEDINGS OF DENTAL AND STOMATOLOGICAL SOCIETIES

FOURTH SECTION

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Harvard Odontological Society

Boston and Tufts Dental Alumni Association

First District Dental Society of the State of New York

Metropolitan District of the Massachusetts Dental Society

Harriet Newell Lowell Society for Dental Research, of the Harvard University Dental School

San Francisco Society for Dental Research

Colorado Society of Periodontists

American Academy of Periodontology

New England Society for the Promotion of Dental Research

William Jarvie Society for Dental Research, of the School of Dentistry of Columbia University

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ORAL DIAGNOSIS[1]

JOHN OPPIE McCALL

Buffalo, New York

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I. INTRODUCTION

The subject of oral diagnosis has been so widely discussed in the last few years, that it would seem impossible to add anything which could justify the presentation of additional papers on this subject. That such papers are in demand in dental societies indicates, however, that the problem is still far from being solved to the satisfaction of all concerned.

There are still some dentists and physicians who give little or no credence to the theory that systemic disease may be caused by oral foci of infection. By far the greatest number in both professions accept this theory, however, at least in an academic sense; but this latter group exhibits wide differences of opinion when the application of this theory is required. The first group denies what it sees with its own eyes. They observe radiolucent areas in radiograms and deny that these indicate a state of disease. If they admit a state of disease, they belittle its local importance, and deny its influence on the rest of the body. Whether or not they accept the interpretation of experiments in animal inoculation, or the clinical evidence of the causal relationship of these infected regions, the fact remains that they condone the presence of disease in the one part of the body, the preservation of whose health has been intrusted to their care. I do not hope to convert those who have withstood the cumulative evidence of the past ten years. To those, however, who are convinced that focal infection can and does produce metastatic disease, I address this paper, with the hope that it may strengthen them in the views they have accepted and provide a practicable means for the application of those views to the patients who consult them.

[1] Read at a meeting of the Pathodontia Section of the First District Dental Society of the State of New York, at the New York Academy of Medicine, October 16, 1922.

Those who seek to cloud the issue by citing cases of incomplete or mistaken diagnosis, barbing their shafts with semi-humorous quips, are indulging in a dangerous sophistry, which may react not only on themselves but on the entire dental profession.

Let me, at the outset, make it clear that while I am firmly convinced of the fundamental soundness of the focal-infection theory, and while my application of that theory may often appear to place me in the ultra-radical group, I do not hold a brief for "100-per cent vitality" of pulps. I believe that it is both theoretically and practically possible to remove the dental pulp and fill the canal with safety to the patient; but I also believe that the best method of accomplishing this has yet to be devised. Furthermore, I believe that comparatively few of the teeth that have been devitalized, and filled by methods used in the past, are free from infection, although the work of a few operators has been sufficiently successful to show the possibilities in this field. I propose, in this paper, to discuss the question of focal infection from three viewpoints: first, consideration of the theory and its application in practice; second, a review of my own personal experience with focal infection; third, a statistical review of the results of extraction of teeth for patients in my practice.

II. A CONSIDERATION OF THE FOCAL-INFECTION THEORY AND ITS APPLICATION IN PRACTICE

It seems well to consider briefly the rationale of the focal-infection theory, before proceeding to its application. The diseases of the human organism have in the past been studied largely from the standpoint of acute disorders; but for many years chronic-disease conditions, of varying degrees of severity, have been claiming more and more of the attention of the medical profession. The etiology and methods of treatment of the acute diseases, of which typhoid fever will serve as an example, have been established for some years. Until recent times the study of chronic diseases has been carried on as a sort of offshoot from the apparently major subject; but we have at last arrived at the point of a fairly clear division as between chronic and acute diseases. While there is no real line of demarcation between the two types, and while chronic diseases may often exhibit acute exacerbations, nevertheless the methods of study, diagnosis, and treatment of the two groups are quite distinct. This has been rather generally overlooked, a fact which serves to explain in part the confusion centering around our present subject.

Acute infectious diseases very commonly exhibit a primary focus which is the seat of the major disorder, and secondary regions or foci of minor

disturbance. Disorders of the nervous system, exhibiting insomnia and mental wandering even to the point of delirium, are common. Neuralgias in various parts of the body are likewise frequent. The heart and circulatory system are often attacked, as likewise the kidneys. These secondary phenomena are so common as to excite no comment on the part of either physician or patient, and are accepted as natural sequelae of the primary disease. It is therefore only logical to expect secondary reactions to occur after the inauguration of chronic-disease conditions. Yet in this field we find a wide divergence of opinion, the reasons for which are not far to seek. The first reason is probably the relative severity of the secondary disease as compared with the mild character frequently exhibited by the primary disorder. The second reason lies in the length of time which frequently elapses between the incidence of the primary disorder and the onset of its secondary sequelae, as also the observation of many cases in which secondary disease has not as yet made its appearance. Another reason for skepticism has to do with the diversified activities of an organism, the streptococcus, which seems capable of producing one disease in one subject and another disease in another. This organism, because of the varied secondary effects which it produces, goes contrary to the laws that govern the activities of most pathogenic bacteria. Such organisms as the typhoid bacillus, the bacillus of tuberculosis, the spirocheta pallida—each produces its characteristic disease and none other. The streptococcus on the other hand attacks the heart in one subject, the kidneys in another, the nervous system in another, and so on through a long list.

There appears to have been much study of the results of streptococcal infection as exhibited in diseased tissues, but little attention seems to have been paid to the mild character of the early reactions to the introduction of this organism into various parts of the body. When we compare the reaction of the body to streptococci, with the reaction to staphylococci, we notice a striking difference. Staphylococci, immediately upon their entrance into the tissues, call forth a sharp response. The defensive forces of the tissues are aroused to their fullest extent, and a most active effort is induced to throw off the invaders, an effort of which the patient is very much aware. On the other hand, streptococci launch a more insidious attack; their entry into the tissues does not call forth so violent a defensive reaction, and the patient is usually entirely unaware that infection is taking place, if it is a pure streptococcal infection. It is only after actual injury to a secondary tissue has resulted that subjective or objective symptoms make their appearance. While the fundamental phenomena of both acute and chronic types of infection are of a similar character, their manifestations

are so dissimilar that the study of chronic disease must usually be carried on in quite a different manner from that of acute disease. The student of chronic disease must be prepared to accept a hypothesis which would not be sound when applied to acute disease.

Let us take as an example Rosenow's experiments in elective localization. These experiments have been criticized because of the large doses of organisms used to produce the secondary lesions. These lesions could be produced in an experimental animal in only two ways; first, by maintaining in the animal a primary focus of infection for a period of time corresponding to the proportionate period necessary for the development of secondary disease in the human subject. This method Rosenow has lately used, producing chronic foci at the root ends of teeth in which small amounts of infected broth were sealed. The second method is by the introduction, into a healthy animal, of a sufficient number of organisms to produce *at once* the secondary disease. That the second method is not illogical is due to the characteristic of streptococcus previously referred to, namely, the unique character of the infection as indicated by the mild reaction it excites. In other words, the normal animal can quickly destroy a smaller number of organisms, when injected into its blood-stream, in the absence of a continuing source of infection located in some primary focus. Even when the so-called massive doses of bacteria are injected, the animal frequently makes a recovery, indicating that the body defenses are not seriously impaired, as is the case when a continuing effort has been made to cope with infection proceeding from an established focus.

Infecton by streptococcus, while thus presenting certain unique characteristics, is subject to the laws that govern all infections. No organism can produce disease, except when resistance to it is lowered. Organisms may live and maintain themselves in the tissues for varying lengths of time without producing disease. It is this factor of resistance which explains the presence of many regions of alveolar infection in individuals who maintain a state of health for long periods of time. It is also the factor of resistance which furnishes an explanation for the apparently paradoxical and gross secondary effects of minute regions of infection at the end of tooth roots. The adjudication of the part that a periapical or parietal lesion may play in the health of the individual is dependent, not so much upon the extent of the lesion as upon the evaluation of the patient's resistance, as indicated by available diagnostic tests.

Among writers on this subject it is common to find reference to arthritis deformans as being the typical disease produced by focal infection. While it is true that focal infection may produce joint lesions, it has been my

experience, and that of the physicians with whom I have been working, that a majority of the patients who are subject to metastatic disease have no joint lesions whatever. The most common site of attack on the other hand is the nervous system. Irritability, psychosis, vertigo, neuralgia, neuritis, neurasthenia, insomnia, and excessive drowsiness: one or more of these conditions is to be found in the greater number of patients suffering from infectious foci. Hence I would suggest that the continued reference to arthritis as being the characteristic focal-infection disease is misleading.

In considering the diagnosis of oral disease, we should first bear in mind that, because of the variations in resistance exhibited by different individuals, no hard and fast rule can be laid down that would be universal in its application. What may be safe for the young healthy patient, may be very unsafe for the elderly, or for the patient having well developed indications of susceptibility to infection. The decision on the latter point can be properly made only after examination by a competent physician. We must be extremely careful, however, that our attitude shall not be overlenient toward teeth with well defined regions of alveolar infection, even if the latter should be small. It is unwise to call upon the body defenses to contend with a continuing infection, even at an age when they seem entirely adequate to the task. It is impossible to forecast the period in life at which these defenses will fail.

For the patient who is ill and has been certified by his physician as suffering from a disease caused wholly or in part by focal infection, the case is quite different Here we must be most critical of the condition of the mouth regardless of conditions elsewhere in the body. We should see to it that recommendations are made which will, without question, free the mouth of foci of infection. Unfortunately for the dentist as well as the patient, foci of infection in other parts of the body are frequently difficult to detect. When they are overlooked a considerable burden of odium may be cast upon the dentist who has ordered the extraction of teeth without visible benefit to the patient. It is therefore impossible to promise a patient that he will be benefited by tooth extraction, yet this fact does not justify the retention of infected teeth. Neither does failure from such a cause invalidate our hypothesis.

It is very fortunate that we have at hand a means of determining, with a high degree of accuracy, whether or not foci of infection are present in the system. I refer to chemical examination of the blood, with special reference to the work of R. N. De Niord, M.D.[2] He has brought out the

[2] See the paper by De Niord on this subject, in this issue—p. 435.

fact that focal-infection cases invariably exhibit an abnormally high content of uric acid in the blood. Patients whose blood shows this uric-acidemia are not only benefited by the removal of foci of infection, but the completeness of the elimination may be gauged by the lowering of the uric acid content in the blood. When all foci have been eliminated, the proportion of uric acid returns to the normal.

The use of this test in many cases, extending over a period of several years, has made it possible to standardize procedure in these infection cases to a considerable extent. In its absence it is necessary, of course, to warn our patients that, in addition to infections in the jaws, they may be subject, also, to infections of the sinuses, tonsils, gall bladder, appendix, and other structures. They must also be made to understand that improvement in their condition is not to be expected through the removal of one source of infection alone. Too often has a patient, after the extraction of two or three teeth, become so discouraged as to refuse to submit to the removal of others equally infected, or of tonsils, etc. It is the part of wisdom, therefore, to make no distinction as to the degree of culpability of any teeth which are considered to be infected, when recommending extractions.

The decision, as to whether a given tooth has become the site of an established infection, must be based upon the clinical examination, pulp vitality test, and the radiogram, plus the history of the tooth. Pulpless teeth, or those giving evidence of loss of pulp vitality or even of advanced pulp degeneration, are the common subjects of established infection. When radiograms of teeth falling in one of these three classes exhibit even slight periapical abnormality, the diagnosis of infection may be considered positive. This involves a distinction between these and the teeth exhibiting periapical abnormality due to traumatic occlusion. In the latter class, evidence of vitality in a pulp, with satisfactory response to thermal and electric tests, renders the case one for treatment rather than extraction. Such lesions may produce a bulging of the periodontal lamella or rearrangement of the alveolar trabeculae, and thus simulate very closely the lesion of established infection.

My present contribution to the interpretation of radiograms lies chiefly in calling attention to the importance of periapical lesions other than the classical circumscribed radiolucent region. For some years radiolucent regions at the root end have been sub-divided into circumscribed and diffuse. A circumscribed region, as usually interpreted, has a more or less symmetrical curved outline of dense bone. The diffuse region may or may not have

an outline of dense bone, but its borders are irregular; and, if there is dense bone, it is usually but slightly marked. This type of lesion presents all gradations of radiolucence from that which is readily distinguished, to that which is differentiated with difficulty from the normal bone around it. Attention should now be called to a third type of lesion, which exhibits neither radiolucency nor radiopacity immediately at the apex, but which does show a definite alteration in the cancellation of the bone adjacent to the apex. The regions referred to may show a somewhat lessened density to the ray, but frequently are only to be detected by the tendency of the cancellous spaces bounding them to assume a somewhat chain-like continuity. In addition to the three types of lesions just mentioned, the periapical radiopaque region, when of a diffuse character, is also to be considered in most cases as indicative of an established infection when occurring around a pulpless tooth, and even when occurring around a vital tooth under certain circumstances. This type also has been much neglected.

These four lesions are the important ones in the diagnosis of periapical foci of infection. It remains to call attention to those parietal lesions which may call for tooth extraction, in cases of metastatic disease. Contrary to the opinions set forth by such investigators as Hartzell and Pollia, it is my belief that the suppurative pockets in cases of periodontoclasia are of relatively less metastatic importance than are periapical lesions, notwithstanding the larger areas of tissue involved. This statement requires qualification with regard to the depth which the pocket has attained, and the evidence of bone involvement beyond the immediate vicinity of the pocket. I do not wish to be understood as condoning the continued existence of ulcerating tissue in the mouth; but I believe that the patient's interests are best served if the choice for extraction falls on a tooth having a slight periapical lesion, rather than upon a tooth having a pocket extending, for example, a third of the length of the root. This opinion rests upon two factors. The first is that of prognosis, which is so much more favorable for a tooth with a vital pulp and a parietal pocket, than for a pulpless tooth. The second reason has already been stated as centering in the apparently greater injury produced by the periapical lesion. The reason for the difference in the systemic effects of the two types of lesion seems to lie in the different modes of drainage. The entire drainage of the periapical lesion is into the blood and lymph channels, whereas the parietal pocket, especially if not too deep, drains largely into the mouth.

If a pocket extends to the apical third of the root, the prognosis should be adjudged with the greatest care. Such lesions are quite apt to have

involved the pulp circulation. Diagnosis of this latter condition is confirmed, if the pericementum at the apex is perceptibly thickened, or if the periapical bone exhibits a diffuse radiopacity. Marked radiopacity of the cancellous bone just beyond the periodontal lamella is usually indicative of a deep seated infection. This is occasionally seen in the absence of pocket formation. The type of case here referred to is one in which the marginal gingiva exhibits a pronounced thickening, with more or less congestion.

Another frequently occurring site of established infection is the impacted tooth. This is usually a third molar, which may be impacted against a second molar; or if on the lower jaw, may be impacted in the ramus. The cuspids are however frequent offenders. The evidence of infection is usually to be found not in periapical rarefaction or in pocket formation, but in rarefaction of an abnormal extent around the crown of the buried tooth, usually involving its pericementum in a lesion evidenced by an abnormal thickness. Another indication in these cases is an alteration in bone cancellation similar to that often found around other infected teeth. This consists of an almost continuous chain of cancellous spaces lying parallel to and more or less completely encircling the periodontal lamella. Even in the absence of either of these signs, the impaction of a third molar, against a second molar or in the ramus, is to be considered as leading almost invariably to the establishment of infection. Partially erupted third molars invariably exhibit more or less infection, which is frequently corroborated by the radiogram. The proof of focal infection in the case of impacted third molars is nearly impossible to obtain by cultural methods. Many cases are to be found, however, in which other factors have been eliminated, and in which secondary disease is only relieved by the removal of these teeth. A somewhat extensive experience in this field leads me to make an almost invariable recommendation for their extraction; or, in occasional cases, the relief of the condition by removal of the second molar.

Root tips are frequently found remaining in the jaws as results of fractures during extractions. There is usually exhibited a varying degree of radiolucence in the surrounding bone. Even without this, the very nature of the operation responsible for their presence makes infection inevitable. Their removal, together with the eradication of other foci of infection, is always indicated.

One other type of primary focus of infection remains to be mentioned, and a most important and much neglected type it is, namely, residual infection. The name "residual infection" means an infection remaining

in the jaw after the removal of a tooth. Much infected tissue may be left in the jaw when a tooth is extracted without subsequent curettage. Even when curettage is apparently complete, small masses of infected bone may still remain. These infected masses are frequently cleared up through the regenerative activities of the body; but often they persist for many years, and, when so occurring, are to be considered true foci of infection. In this field, as in the case of impacted teeth, evidence is frequently to be obtained only through clinical observation after elimination of other foci. The use of the blood-chemistry test, revealing a continuing high content of uric acid in the blood, frequently justifies operation upon such cases, whose radiographic diagnosis was occasionally indeterminate.

III. A REVIEW OF THE AUTHOR'S PERSONAL EXPERIENCE WITH FOCAL INFECTION

Realizing from conversations with many dentists, that personal experience with focal infection provides striking first hand evidence of its effects, I have felt that a brief description of my own experience with metastatic disease might be of value. In December, 1920, I found myself subject to recurrent attacks of conjunctivitis. To this was added a pronounced erythema nodosum. Chemical examination of the blood showed a content of uric acid slightly above normal, and absence of adrenalin, but no indication of kidney damage. I had at that time four pulpless teeth, which showed little or no indication of periapical lesions, and a lower third molar with a vital pulp but with recurrent pulpitis. These five teeth were extracted with prompt subsidence of the secondary symptoms. In June of the following year, I developed a very pronounced asthenia, evidenced by extreme lassitude, especially upon arising. There was no recurrence of the skin or ocular disorders, but a persistent myositis of the deltoid muscles developed. Blood tests revealed a larger content of uric acid than at the first examination. My tonsils were slightly infected and a lower first molar gave evidence of pulp degeneration. The molar and tonsils were removed, following which a marked improvement occurred. Blood tests the following January showed a return of the content of uric acid to the normal level. In July of this year (1922) there was a recurrence of the erythema and conjunctivitis, with some asthenia, also lymphatic involvement on the left side of the neck. No blood test was made, but mouth examination gave evidence of pulp infection in two lower molars. These were extracted with prompt improvement. The favorable reaction, which in

each instance has followed the removal of infected regions, has been the most convincing argument on the sound relationship of these foci to the systemic disorders.

IV. A STATISTICAL REVIEW OF THE RESULTS OF EXTRACTION OF TEETH FOR PATIENTS IN THE AUTHOR'S PRACTICE

It remains to outline for you the results of the removal of foci of infection for those who have been referred to me for oral diagnosis. It has been my good fortune to be able to follow a large number of these cases, and to find out wherein lay the responsibility for occasional failures to obtain benefit. The following is a summary from my case records:

In a little less than five years I have taken radiograms for nearly 4,000 individuals at the request of physicans and dentists, and in course of the routine of my own practice. Of these, 1,173 have had the radiograms taken for the purpose of locating foci of infection with the object of securing relief from physical ailments. Among these patients there has occurred, to my belief, every type of disease which has been credited, by medical authority in whole or in part, to focal infection. I have been able to secure a trustworthy report on the results obtained by the clearing up of foci from 654 of these cases, including in this group no cases coming to me later than July 1, of this year—results in such recent cases are uncertain. I have subdivided these 654 cases into two groups, in one of which elimination of foci has been complete; the other, in which it has been incomplete. In the "complete" group, which consists of 503 cases, all foci which could be diagnosed by medical and dental examinations were removed. This process included tonsillectomy in a large percentage of cases, and several appendectomies. Of the 503 cases in this group, 419 were reported as obtaining satisfactory results, 78 secured definite improvement in their condition, 6 cases were not improved. There were 6 cases in this group which were complicated by other diseases, such as advanced diabetes and nervous disorders, which fell either in the group of failures or of partial successes. The percentage of success in 503 cases is thus seen to be 83, with an additional 15 per cent of partial success. Some of the cases of partial success showed marked improvement soon after operation, with a partial relapse at a later date. Other cases took a long time to build up to an entirely satisfactory condition of health. There may be thus a certain shifting in these figures. Nevertheless, the percentages remain at essentially the same levels.

The group receiving incomplete treatment includes those in which the patients for various reasons failed to have all indicated foci removed. In this group 86 cases showed no improvement, 53 cases were relieved in varying degree and 13 cases were considered as definitely successful. Among the 86 unsuccessful cases, several showed more or less improvement following operation, with subsequent recurrence of the original condition. Among the 52 partially successful cases, there was also some fluctuation. It is worthy of note that the cases in which good results were obtained, upon incomplete elimination of foci, were patients whose ages were below 40; or whose indicated foci, whether teeth or tonsils, exhibited but little evidence of infection. The percentage of total failure, due to incomplete elimination, is 57. The percentage of partial success was 34, while the successful cases give a percentage of but 9.

I might state that I have under observation several patients in whose mouths there are different numbers of teeth giving radiographic evidence of infection, who seem at present to be entirely well. These are not listed in the foregoing tabulation. Neither have I listed many cases sent to me, for radiograms, by dentists with the statement that the patient was in poor health. I have preferred to list only those cases in which a diagnosis of focal infection has been made by a physician.

It must be remembered that, in a great many of these cases, the use of chemical tests of the blood gave the clue to the incomplete elimination of foci and resultant failures in treatment. Cases thus managed, upon further search for and removal of foci, gave the desired improvement; and the high percentage of success, shown for cases in which elimination was complete, is to be traced largely to this invaluable diagnostic aid.

In the light of these findings, it is evident that, while we are not to abandon our efforts toward the solution of the pulp-canal problem, we must be very critical of the work of the past; and, in diagnosis, we must be suspicious of every possible focus of infection as long as a patient's health gives evidence of injury from that source. Our slogan need not be "100-per-cent vitality," but it should be "100-per-cent freedom from infection."

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DISCUSSION OF THE FOREGOING PAPER, BY JOHN OPPIE McCALL, ON "ORAL DIAGNOSIS"

By E. E. Smith and M. L. Rhein, of New York City; and Dr. McCall

Dr. E. E. Smith (New York City): Dr. McCall's most valuable paper presents two questions of particular interest.

(1) First, *What symptoms may be attributed to oral focal infections?* Most obvious, and most definitely lending themselves to a reasonably positive conclusion as to the rela-

tionship, are symptoms arising from frank metastatic foci or from inflammations set up by absorption products. These secondary manifestations, whether they arise in the joints, in the endocardium, in the eye, or in some other region, strongly suggest the periapical focus, where such has been demonstrated to exist, as the primary factor. Even here, however, a considerable degree of caution is always to be observed, since for example it is quite generally true that a case exhibiting periapical foci will likewise show a similar streptococcal infection of the tonsils. Indeed, it is probably true that bronchial infections with streptococcus viridans are as frequently (and perhaps even more frequently) derived from the tonsils as the primary focus, as that the *periapical* focus is the initial fault. On the other hand, where the eye is involved, we more frequently find that the teeth are *primarily* at fault, even though the tonsils carry the same infection. Nor may we overlook the sinuses as a menacing source from which in many cases secondary streptococcal reactions arise. Finally, it is to be borne in mind that the intestinal tract is a frequent carrier of streptococci, and that the possibility of this primary origin of streptococci (or even other sources not enumerated) must always be considered. Nor is the differentiation always easy or even possible. Dr. McCall recognized all this, and I am merely adding emphasis to what he has already said, in again calling attention to this matter.

When, however, we come to less definite evidences of secondary foci or effects, symptoms consisting of pain without gross inflammatory changes, and especially symptoms consisting of hyper- or hypo-function, such as the list of symptoms of the nervous system to which Dr. McCall has referred, the relation of such manifestations to periapical foci or even to any foci at all, as such, presents much greater difficulty. We know that the nervous system manifests itself to a considerable degree through activities that are under the influence of the endocrine secretions. Consider for example the exhibition of irritability, psychoses, insomnia, in fact the train of so-called neurasthenic manifestations to which Dr. McCall has referred. These may be brought about in a number of ways. They are the train of symptoms that are observed in some cases of hypopituitary function, such as may occur in tuberculosis of this gland, for example; and when so brought about are relieved by the administration of pituitary substance, as I have observed. In such a patient, periapical foci may exist, yet the symptoms may be neither relieved nor ameliorated by eliminating such periapical foci. It is because of these possibilities that Dr. McCall has wisely cautioned against too favorable a prognosis when the teeth lesions are removed by extraction and treatment.

Another possible cause of this same train of nervous symptoms is found in some intestinal toxemias. It has long been recognized that in certain individuals intestinal putrefaction, when excessive in degree, is associated with this train of nervous manifestations. Recent years have added to our knowledge of this subject by revealing that, in some instances, the products thus formed are identical in pharmacological action with certain of the endocrine active principles, and can supplement or unbalance the action of the latter upon the nervous mechanism. Thus, tyramin, through bacterial action may be produced from the well known amino acid tyrosin (formed in digestion) and, when absorbed, produces the same action upon the sympathetic nervous system as does epinephrin, an active principle of the suprarenal glands. Likewise histamin, produced by bacterial action from the amino acid histidin, exerts an action upon the nervous system and is even capable of producing the symptoms of *anaphylactic shock*. To be sure, under ordinary conditions the system is protected from these various effects by deamination, by oxidation, or by conjugation of these products in absorption or by the liver; but that, in spite of these natural defences in certain cases, products of this kind do gain entrance into the system, and pro-

duce toxic nervous symptoms such as have been cited, cannot be denied. Indeed, this factor must always be recognized and properly verified in reaching any conclusion as to the cause of the nervous symptoms we are considering.

From the foregoing, it is clear how uncertain is any positive conclusion of an etiological relation of periapical foci to a symptom manifestation in an individual case, notwithstanding that it is well established that such relation *does* exist, and is a common occurrence when we are considering cases in general. The possibility, and even a considerable degree of probability, is beyond question in many instances. That the relation of cause and effect does in fact exist, in an individual case, requires more than the recognition of the symptoms and detection of the foci; it requires the weighing of all other possibilities, and either their exclusion or at least the *recognition* of these possibilities.

Dr. McCall has called attention to the diversity of activities of streptococcus. I can fully endorse what he has said in respect to this organism. I must differ with him in his view that the tubercle bacillus or the organism of syphilis produces its characteristic disease and none other. Rather, I would mention both of those infecting agents as examples quite similar to streptococcus in capacity to produce varied secondary effects. In the case of syphilis, the secondary effects are even more obscure than those of streptococcus; for it may be a matter of years after the establishment of the infection, early in which interval the primary focus has entirely healed and disappeared, before tertiary manifestations, involving heart, kidneys, nervous system, and so on through a long list of organs, give symptoms of their involvement. Indeed, we can say with almost any infection, that it may give rise to secondary manifestations and that it is good fortune when it does not. I would regard streptococcus, in its production of varied secondary effects, not as going contrary to the laws which govern the activities of most pathogenic bacteria, but rather as conforming to the general rule that bacteria do or may give rise to secondary effects. Indeed, in this conformity, we have a stronger support of our present views of the behavior of streptococcus than if the organism worked contrary to the general rule.

Of considerable interest and importance is Dr. McCall's reference to the conclusion of Dr. DeNiord, that focal infection cases invariably exhibit an abnormally high content of uric acid in the blood and that the completeness of the elimination of such foci may be gauged by the *lowering* of the percentage of uric acid in the blood, complete elimination being accompanied by a return to the normal. I must confess that this view is quite new to me and that I feel inclined to scrutinize the proposition from several angles.

It has been the view in the last few years, of some of the workers in this field, that a high content of uric acid in the blood is to be attributed to a retention from the loss of renal function, whereby the elimination of this waste product is incomplete; and, further, that it is ordinarily one of the very early signs of loss of renal function, preceding very decidedly the retention of urea, and even more decidedly of chloride and creatinin retention. If then, it be true that a high content of uric acid in the blood is due to diminished renal capacity to eliminate this product, foci of infection would of necessity exert their influence (upon the amount of uric acid in the blood) *only indirectly*, through bringing about loss of renal function. That focal infection may do this, or *tend* to do it, is highly probable. However, *any other cause* of loss of this renal function would also tend to the same result. I hardly think Dr. De Niord would care to state that infective foci are the *only* cause of these early renal changes. Moreover, if it is to be concluded that removal of the offending foci would restore the content of uric acid in the blood to normal, it must likewise follow that it would of necessity lead to the restoration to normal of this renal

function. I hardly think Dr. De Niord would care to subscribe to this as a universal rule. In other words, Dr. McCall's conclusion in regard to the content of uric acid in the blood, based upon Dr. De Niord's statement supported by his own observations (of an invariably high content of uric acid in the blood with foci, returning to normal upon their removal), would, in our own experience in this field, be modified to the extent of saying (a) that foci of infection may produce sufficient toxic effect to lead to loss of renal elimination of uric acid in which event such foci would be accompanied by a high content of uric acid in the blood; and (b) that, if the foci be removed before the loss of renal uric-acid eliminative function is permanently established (presumably by initial organic renal changes), then, in that event, the removal of such foci might lead to restoration of a normal content of uric acid in the blood. In the event of *permanent establishment* of such organic renal changes, however; and, further, in the presence of *other* causes of loss of renal uric-acid eliminative function, which have not been removed coincidently with the removal of the foci—in *such* event, the renal uric-acid eliminative function would not be restored and the high content of uric acid in the blood would not return to normal. Nor in that event, would the conclusion be justified that all foci had not been removed. Other foci might be present, or causes other than bacterial foci might determine the high content of uric acid in the blood; or again, the renal function might be permanently impaired, thereby preventing the restoration of the content of uric acid in the blood to normal.

It is indeed rare that a pathological condition such as we are considering is so limited in its etiology, and when once established is so thoroughly corrected by the removal of a single cause or kind of cause, that we may anticipate the invariable return to normal when such cause has been removed. It would be much simpler were it so, and we would avoid many times the baffling problems that confront treatment. However, this does not in any way render valueless the observations on the content of uric acid in the blood, made by Drs. De Niord and McCall; but it cautions us against a too dogmatic application of the valuable relationship which they have undoubtedly observed in many cases.

In Dr. McCall's own case, which has been related, it seems that, after the removal of four pulpless teeth (which showed little or no indication of periapical lesions) and of a lower third molar with a vital pulp but with recurrent pulpitis, he was relieved of attacks of conjunctivitis to which was added a pronounced erythema nodosum and a content of uric acid in the blood slightly above normal. These manifestations may well be the expression of a streptococcal toxemia. It would be interesting to know if Dr. McCall determined the nature of the organism that was active in the pathological condition of the eye. If his teeth, with their relatively slight periapical infection, were the primary etiological factor, it is remarkable that a myositis should have developed from the same cause within the short period of six months, since the teeth were doubtless freed from any existing infection and the time elapsed seems quite too short for the teeth ordinarily again to establish foci sufficient to cause the asthenia and myositis. The third outbreak a year later, when there was a recurrence of the skin and ocular disorders, still further suggests either a remarkably unusual susceptibility or that the teeth foci were in fact not primary but secondary to some other foci possibly sinus or even intestinal, and that these latter had been allowed to remain to produce the recurrences noted. So far as my observations extend (and probably many of you have had greater opportunity to make similar observations): where the teeth infections are the primary fault, their removal will not be followed by recurrence in such short periods of time.

(2) The second important question presented by Dr. McCall's paper is this: *With oral foci of infection in a case without symptoms, when, if ever, may treatment be conducted*

without extraction? Dr. McCall has touched rather lightly upon this phase of the subject, practically limiting himself to an acknowledgement that it is possible to remove devitalized dental pulp and to fill the canal with safety to the patient. It is a question of very great moment. I need not remind you that in many and indeed most cases extraction means a permanent reduction in efficiency of mastication, which is to be avoided, if it may be done with reasonable safety. In an individual of robust constitution, without symptoms, it is certainly rational to give this matter earnest consideration. I have been much interested in the reactions of patients to tests by intradermal injections of suspensions of the patient's own infecting organisms, as conducted by one of your members, Dr. H. J. Kauffer, as an indication of the patient's established immunity and hence as a guide to judgement in the matter of extraction. It seems to me that it is along such lines that the future is going to enable you to base your decisions on more than general and necessarily vague considerations.

It is a great accomplishment to remove primary oral foci and relieve systemic disease. It is likewise an obligation to avoid unnecessary reduction of efficiency. In the satisfaction of accomplishing the one, I am sure you are mindful as well of the other. It is not alone the pathology that must be considered but the patient. The time-worn advice, to treat the patient and not the disease, is quite as applicable in dentistry as it is in medicine.

Dr. M. L. Rhein (New York City): It is a pleasure to corroborate Dr. McCall on the fact that many systemic diseases are caused by oral infections. This is not a new position for me to take. Thirty-five years ago I commenced to endeavor to impress the knowledge of this fact not only upon the dental but upon the medical world, and have never ceased my efforts in that direction. During those early days I was considered a radical of radicals. While I have in no way modified my views, today I find myself classed among the conservatives because I still claim to be able to treat pulpless teeth in such a way as to prevent them from being a menace to the health of the patient.

Dr. McCall's paper is an admirable one in many respects. The different roentgenographic pictures of periapical conditions are not only well described but, what is more to the point, are truthfully described. Dr. McCall becomes somewhat paradoxical when he speaks of nothing but extraction as a remedy for focal infection, and yet goes out of his way to disclaim membership in the "100-per cent club." It is easy to sympathize with his point of view when one considers what average root-canal therapy means. The value of teeth for the purpose of mastication is such that we cannot believe there are no rational methods of preserving them in a state of health and usefulness. This general principle, however, has been made a pretext for root-canal treatment of such a nature as simply to encourage the spread, in individual cases, of any toxins that may have been formed.

Beautiful as is the histologic character of Dr. Davis's work on the filling in of the canals of a few isolated cases, with cementum, it is unthinkable that these few cases would be used as a basis for imperfect root-canal therapy. The same criticism may be made of other methods. The unfortunate thing about root-canal therapy is that it requires infinite patience, and so much time and expenditure of money as to render it an economic impossibility for many. A makeshift, however, will not supply this deficiency. We can only hope for the presentation, in the future, of a therapy that will more quickly place such teeth in an absolutely dependable condition as far as health is concerned.

It is a great pleasure to be able substantially to agree with Dr. McCall in his position on the difference between the effects on the system of focal infection and general oral

sepsis. Let us for a moment dissect this differentiation, because the two are generally classed together by a majority of dentists and physicians. A dental focal infection is a limited infected region in the dental tissues, which never reaches a state of suppuration and from which there is no drainage. The very diminutive character of the focus makes it a most dangerous form of infection, because toxins are formed which continually pass into the circulation, and yet suppuration is impossible. The stage of suppuration, in infections, is nature's curative method for producing drainage, and in this way leaving an escape from the continuous absorption of the toxins. I take my stand squarely with Dr. McCall on this issue. While recognizing the enormous harm that open suppurative conditions in the mouth produce, yet their danger to the individual is almost insignificant when compared to that of the granuloma sealed in the periapical region.

A very interesting and instructive feature of the paper is the part referring to blood chemistry. Perhaps the most important point in the paper is the statement that a largely increased content of uric acid in the blood is an indication of dental focal infection. In the early '80s Pierce first announced that excess of uric acid in the blood was the etiologic factor in dental periclasia. For some years this view was held by a large number of men. This theory was exploded soon afterward, when it was discovered how frequently such an excess of uric acid manifested itself in different types of malnutrition.

In the September number of the *Dental Cosmos* (1922), there is a very interesting article on this subject by Dr. J. A. Toren. Dr. Toren claims that, in serious cases of dental focal infection, there is invariably present a very large, peculiarly shaped, white blood corpuscle, which is not found in any other form of infection.

We must, for the present, accept such declarations with a grain of salt. If either or both statements were true, it would be a very welcome fact. It would at once solve all the uncertainties of periapical infections. It would be a truly wonderful advance in scientific therapy. It may be true that one or both of these results can be obtained in different types of streptococcal infection, but that they are characteristic of periapical infection is a remarkably strong assertion. Both of these signs in the blood, if substantiated, are of enormous value in regard to the prognosis of teeth contiguous to a region of focal infection. In all such matters, proclamation of findings requires substantial corroboration.

In one part of Dr. McCall's paper he speaks of experiments, by Rosenow, in which chronic foci of infection were produced at the root ends of teeth in animals. In a series of experiments on over half a dozen dogs, in collaboration with Dr. Gies, we failed to produce a chronic streptococcal infection at the root ends in these dogs. Whether this would be possible in the case of a dog in which disease decreased its resistance, I cannot say. At least thirty years ago I tested this matter on six guinea pigs by injecting into the alveolar structure broth that was most highly infected with streptococci. The healthy pigs threw this off within thirty-six hours, showing no effect either locally or systemically, while three of the pigs, that were tubercular, died within forth-eight hours from the effect of a similar injection.

There is one important feature of this particular division of oral diagnosis that I fear Dr. McCall has overlooked. Teeth in which the pulps are still alive frequently are found to contain infected pulps. The study of diseased conditions of the pulp is still in its infancy, and the diagnosis of them is generally overlooked. In one of my articles on this subject, written many years ago when I was known as a bold, bad radical on this subject, I unhesitatingly advised the immediate removal of all pulps of this character. This has been my invariable practice, relying upon my ability to handle the root-canal treatment

successfully. Nothing is more unfortunate than to nurse teeth of this kind in the jaw; and the present wave of fear of root-canal therapy, and feeling of safety as long as the pulp shows signs of vitality, is responsible for an unwarranted amount of dental focal infection that has been very much overlooked.

It is impossible for me to close these remarks without uttering a protest against the use of the general title "oral diagnosis," which I believe is the title of Dr. McCall's paper, and which is used so much by men who assert the claim to being specialists in oral diagnosis. It is unquestionably correct that the subject matter we are considering this evening is an important factor connected with oral diagnosis, but it by no means has a right to be considered all that is indicated by these two words. In fact, it is a question whether what has been left unsaid is not the most important matter connected with oral diagnosis.

That a dentist who is not thoroughly conversant with the pathology of diseases of every part of the body, in other words is not a thorough physician, can materially assist in oral diagnosis is unquestionably true; but, it is equally true, that he is unable personally to fill the rôle of an oral diagnostician. For that reason I think it timely that attention should be called to this misuse of the term. This is no idle quibble on my part, because some day oral diagnosis will come into its own as the most important means of determining the commencement of any serious departure from the physiologic in the human body.

With all the advances which scientific medicine has made in the past forty years, it has progressed very little as far as the diagnosis of disease in its incipient stages is concerned. The physician is generally able to diagnose different types of disease when it has reached such a stage that the case becomes hopeless. That this fact is well understood accounts for the different cults and the wide-spread interest that is manifested in every claim of achievement in this direction. It is a generally accepted fact that the blood contains the key to the condition of the entire body; and that, when part of the machinery is not working up to "100 per cent standard," this failure is reflected in the quality of the blood. If only we were able to read correctly what the blood tells us. It is a well known fact in every form of life, whether human, animal or plant, that as soon as nutrition is interfered with, its first symptoms are manifested at the periphery of the nutritional stream. In the human body this position is marked at the point where the capillary stream enters the veins. We, as dentists, duly appreciate the fact that the extraordinary anastomosis of the capillary system in the gums renders this the longest capillary system in the body, if it could be straightened out. Granted that this is true, it follows as a natural sequel that, in the gums, is to be found the first evidence of defective nutrition. This is not only a fact that has been corroborated by case after case, but the important part of this fact is that every different type of malnutrition leaves a different characteristic sign in the gum tissue, which, when understood, can be read as plainly as any ordinary type. Some time in the future, when every dentist is a thoroughly educated medical man, this will be considered the most important portion of oral diagnosis.

Dr. McCall (in conclusion): Let me, at the outset, thank Drs. Smith and Rhein for their very carefully considered remarks. It is a pleasure to have had so many points of agreement, and also a pleasure to have this opportunity to clear up some of the points of apparent disagreement.

Dr. Smith has called attention to the fact that a high content of uric acid in the blood may be brought about through agencies other than focal infection. This is a point recognized by De Niord, but which I inadvertently failed to mention in my paper. These factors are cachexias from whatever cause, primary anemias, leukemias, and massive doses

of radium and x-ray. He does not attribute it so much to diminished renal function, although this condition is frequently associated with focal infection. In his experience, the return of the uric acid content to its normal level, in cases where focal infection exists, is dependent on the elimination of the foci of infection rather than upon an improvement in renal function, and in fact is to be accomplished only by such means. Concerning the effects of focal infection on the nervous system, my thought was not to gainsay the undoubted effects of other influences on this part of the organism, but rather to add to this list the agency under discussion. The recognition of this relationship may be an aid in diagnosis.

With regard to my personal case, I should explain that the myositis was apparently superinduced by a "boil," but persisted after this acute infection cleared up. The asthenia became manifest after this also. My explanation is that a lowering of resistance occurred as a result of the furunculosis; and a previously existing slight infection in one tooth, and in the tonsils, became either more pronounced or more effective in producing systemic results. It must be remembered that focal infection may exist, without producing clinical symptoms, in the presence of a high resistance either natural or acquired. It is also to be remembered that a present high resistance, demonstrated as by Dr. Kauffer's test, for instance, cannot be depended upon to remain high indefinitely. Hence we must not be too lenient toward suspicious teeth in the mouths of the robust.

Let me corroborate emphatically what Dr. Rhein says about the etiologic importance of the tooth with a vital but infected pulp. Such a tooth may influence the health quite as much as the pulpless tooth. And with safe pulp-canal therapy, and filling, such a tooth may be made safe for its host, providing irreparable damage has not occurred to the investing tissues.

Dr. Rhein's criticism of the use of the term "oral diagnosis" in this paper is well taken. This term should include the recognition of any abnormality in the mouth, whether its significance is chiefly local or whether it may be utilized in the detection of diseases of the general system. I must plead the prevailing usage of the day as an excuse for not specifying in the title the limitation of the scope of this paper.

DISCUSSION OF THREE PAPERS, BY THOMAS P. HINMAN AND HARRY B. JOHNSTON, WHICH WERE READ BEFORE THE FIRST DISTRICT DENTAL SOCIETY OF THE STATE OF NEW YORK AND PUBLISHED IN THE MARCH ISSUE OF THIS JOURNAL, UNDER THE FOLLOWING TITLES:[1]

(I) "The Importance of the Condition of Apical Cementum in the Treatment of Infected Teeth" (Hinman).....pp. 103–108

(II) "The Destruction of Apical Infection" (Johnston).....pp. 109–116

(III) "A Method for the Filling of the Pulp Canaliculi by Diffusion" (Johnston).....pp. 117–128

By Dr. Arthur B. Crane, of Washington, D. C.; Drs. Rodriguez Ottolengui, and M. L. Rhein, of New York City; and Dr. Harry B. Johnston, of Atlanta, Ga.

Dr. Arthur B. Crane (Washington, D. C.): I feel it is not my duty merely to accentuate the good things in the papers and to say nice things about the essayists. You did not ask me to leave my practice and come over here for that purpose. It is my duty, I think, wherever I can, to point out a mistake, or, wherever possible, to add something in the nature of constructive criticism.

Dr. Hinman very wisely expressed his gratification that some have continued, in spite of adverse criticism, to try to save teeth with diseased pulps; and he condemns unsparingly that (perhaps) growing group of men who would extract every tooth that lacks pulp vitality. Of course, we are all familiar with the argument of the men who favor extraction in preference to attempted treatment. Their argument is that the tooth, once the pulp has become devitalized, is dead, and that nature will not tolerate a dead organism. Let us see whether we can offset that argument.

Let us suppose that we have, in the mouth of a patient, a perfect tooth. So far as we can determine, there is absolutely no break in the enamel covering, and all conditions are apparently normal. Not even the most rabid "100-percenter" would condemn that tooth. We have there a live organ, normal, functioning and innocuous. In course of time, caries destroys the enamel and part of the dentin of that tooth. The patient presents; we discover the cavity, properly treat the dentin, and put in a filling, or a metallic restoration. Now we have what? We have a live organ, supporting dead or foreign substance. Certainly no "100-percenter" would condemn that tooth to extraction, if restoration is properly made; and yet we have live tissue supporting dead, foreign, material.

Now, we will go a little further. Through some unforeseen calamity, the pulp becomes infected, and we must remove it. We fill the root canal; and, provided infection has not

[1] It is the policy of this Journal to publish discussions of papers in the issues containing the papers themselves, in order to serve the convenience of all concerned. Unfortunately for our intention in this relation, the first part of the manuscript of this discussion was received for publication on September 23; the last, on December 16, 1922.—(W. J. G.)

progressed enough to devitalize the pulp, and assuming that we have not introduced infection, we have a living organ, supporting dead and foreign substance. The pericementum and cementum are still alive. The dentin becomes, in effect, the filling of the shell of cementum, just as your gold filling becomes a filling of the enamel of the tooth. So let us get away from the idea that a pulpless tooth is necessarily a dead tooth.

I was very much interested in Dr. Hinman's theory of continuing vitality in apical cementum that had been denuded through the process of infection. He expressed the idea that perhaps vitality in the gingival third, and in the middle third, might lend life to the apical third. If he really believes that, after studying histology, he has had access to more modern histology of the cementum than I have. So far as I know, there are no canaliculi in cementum, and there is no way by which nutrition can pass from one part to another; and I cannot see how life can be given to the apical part by any other part of the cementum. I believe the cementum receives its life through the red blood stream, and that, when the connection of the pericementum with its red blood stream is removed from contact at any point, the cementum becomes a dead and necrotic tissue. If this is soaked in the products of infection, I do not believe there is any known method that will restore it to life; to say nothing of health.

Dr. Hinman spoke of determining, by means of the roentgenogram, when cementum was eroded. I do not exactly remember the thickness of the apical cementum, but certainly it is hardly thick enough to show in a roentgenogram. What Dr. Hinman showed was not a destruction of the apical cementum alone, but also of the dentin. I believe there are many cases of destruction of the apical cementum that cannot be detected with the very finest technique in roentgenology.

Dr. Hinman very properly condemns the type of teeth that I classify as Class 2 and Class 3. Secondary or diffused granuloma, and the radicular cyst, certainly place any tooth beyond recovery, because so much dead cementum is present that it can never be restored to life. Therefore, such teeth must be condemned to extraction, unless you wish to resort to root resection.

Although much has been said upon the subject of electrolytic treatment of periapical disease, an almost chaotic confusion of ideas regarding it still prevails. This confusion may be the result of a lax use of terms, of a false conception of electro-chemic phenomena, or of failure to give due consideration to electro-biology. It is unfortunate, therefore, that the essayist should speak of "ionizing" infected teeth in the very first sentence of his really constructive paper. Teeth, whether infected or not, cannot be "ionized." It may weary some, but let us consider fundamentals for just a minute. As is well known, substances consist of molecules and these, in turn, of atoms. It is now universally recognized that the force that holds atoms together is electrical and is accomplished, at least in the recognized electrolytes, by the transfer of one or more electrons from the negative to the positive elements. Thus, zinc, a positive element, is capable of existing in combination with chlorine, which is negative, to form zinc chloride; or potassium, which is positive, is capable of combination with iodine, which is negative, to form potassium iodide. There is an intermolecular and interatomic movement of such elements, even in the solid state; but when they enter into solution, there is a loosening of the attachment of the electrons, called dissociation, by which there is a more independent freedom of movement.

When a compound is capable of decomposition by the electric current it is called an electrolyte. The facility with which an electrolyte may be thus decomposed depends upon several factors, probably the most important being the relative number of dis-

sociated or separated atoms. When there is dissociation, so that the atoms may travel with the electric current, the substance is said to be ionized and the travelling atoms or radicals are called ions. Let it be clearly understood, however, that it is not necessary that the electric current should pass through the solution in order to ionize the compound. All that is necessary is that, by the act of solution, the attachment of the electrons should become so loosened that the elements are rendered capable of travelling with, or being conducted by, the current *if it is* introduced. It is an established fact, however, that not all compounds that enter solution are thereby ionized.

Since a tooth cannot be decomposed by the electric current and cannot enter solution, it is obvious that it cannot be "ionized;" so let us stop the misuse of this word.

It seems to be a popular conception that the movement of ions, with the electric current, is a sort of orderly parade of positive atoms going in one direction and negative atoms going in the other; but, so far as we know, electrolysis is not an atomic movement as such, but a progression of dissociated atoms, or ions, from one molecule to another by the transfer of electrons. The compound of the dissolved electrolyte is always in solution except at the polar extremes of the mass of electrolyte, where it is given off in the form of its elemental components.

Iodine as such is not ionized. It is, therefore, necessary that some substance, which has the power to receive the negative electron from the iodine atom, should be supplied or added to iodine solution in order that the iodine may travel or be conducted against the current. Potassium iodide is the substance usually added for this purpose; its addition makes it possible for the ion, I_3, to travel from the negative toward the positive pole. Churchill's tincture of iodine, or the tinctura fortior iodi of the U. S. Formulary, contains five times as much iodine as potassium iodide. The ordinary U. S. P. tincture of iodine contains approximately three and a half times as much potassium iodide as iodine.

Bearing in mind that it is necessary to have the potassium ion to facilitate electrolysis of the I_3 ion, it would seem that the large excess of iodine in Churchill's solution would not be available for electrolytic medication, after the potassium iodide had all been satisfied. On the other hand, the deficiency of iodine in the ordinary tincture would seem to detract from its efficiency. As between these two iodine compounds, Lugol's solution contains twice as much potassium iodide as iodine, and has been found to be a valuable agent for electrolytic medication of periapical areas. The theoretically ideal solution would contain about three parts of iodine and two parts of potassium iodide; thus the iodine could be electrolyzed and all of the elements satisfied.

It will be obvious, from the foregoing theorization, that Dr. Johnston has made a wise selection in Churchill's tincture, but it is questionable if he has an electro-chemic strength of 19 per cent iodine as he claims; for, as has been shown, it is only electrolytic to the extent to which the potassium content is available and dissociated.

Dr. Johnston stated that he has been able to prove by experiment that the iodine contained in Churchill's tincture will be entirely driven out of fifteen drops of the solution by a current of one milliampere continued for thirty minutes. I hope, in closing the discussion, he will describe this experiment. I have watched men using iodine for electrolytic medication and heard them express the same idea (that the iodine had all been driven out by the current) because the cotton with which the electrode was wrapped entirely lost its brown color. These men overlooked the fact that hydrogen ions were being conveyed by the positive current, and that in this manner the iodine was reduced and rendered colorless. Iodine in many of its chemical combinations is colorless; for instance, in potassium iodide. Iodine in starch or other colloids may be lavender or

purple, while exposure to the atmosphere causes oxidation and blackening. It will be plainly seen, then, that the changing color of iodine is influenced by factors other than electrolysis.

To reason from the antiseptic strength of tincture of iodine to that of the ion, I_3, also leaves very much to conjecture. Thus, the experiments by which Dr. Johnston has deduced the facts upon which he bases these statements should be checked by other investigators.

Regulation of the dosage or amount of current, according to the number of canals, also seems to be based upon incomplete data. The electric current, all other things being equal, flows in the direction of best conduction. The resistance to the current that a very small apical foramen offers may cause the major part, perhaps all, of the current to pass through the canal with the widest foramen. If proof of this deflection of the current is desired, it is very simple to place a double electrode on the cheek or lip, so that one side comes into contact with the moist mucous membrane and the other with the dry skin. The current will be felt to pass through the moist surface.

It is therefore rather arbitrary to regulate the amperage by the number of the canals. A much safer method is to treat each canal separately. Where the opening of the tooth root is in actual contact with the membrane that lines the floor of the antrum, the current causes but little pain and great amperage can be used, because the current encounters practically no resistance. If a triple electrode is introduced into the canals of such a tooth, the two canals that are in contact with alveolar bone will transfer little of the current compared to the canal which is in contact with the mucous membrane of the antrum.

I do not know who it was in the Public Health Bureau that furnished the information relative to the electro-activity of iodine. He evidently misunderstood the import of Dr. Johnston's question, for the relative electro-activity of various ions depends upon their ionic mobility and the extent of their dissociation. The ionic mobility of the mono-iodide ion is about 70, which is about the same as the ionic mobility of chlorine, bromine, SO_4, NO_3, and potassium. The ionic mobility of the tri-iodide ion is about 55 per cent of that of the mono-iodide, and therefore only a little greater than that of sodium.

With an excess of iodine in a mixture, such as Churchill's tincture, there is very great preponderance of tri-iodide ions and it is possible to transport a fair amount of these ions electrically. Of course, the total amount that can be thus conveyed depends upon the number of dissociated ions present, and this is a rather complicated matter.

Dr. Johnston does well to introduce his second paper with the statement that all of our present methods of filling a pulp canal are crude and unsatisfactory. It is hardly conceivable, however, that the perfect filling will ever be discovered, because of, or in spite of, the very laws which the essayist has called into action in his endeavor to improve his results in this field of endeavor. The laws of diffusion, capillary attraction, and absorption, apply only to liquids; and only the relatively insignificant amounts of solids that can be dissolved in a liquid, without changing its consistency as a liquid, can in any way be affected by these natural laws. It is self-evident, therefore, that when a solvent evaporates, diffuses, or is absorbed as the case may be, the amount of solid that remains can in no sense of the word constitute a filling. If we use the word "obliteration" instead of "filling," we shall readily comprehend that there is a wide difference between the penetration of the canaliculi and tubuli with a material that may outline them, and the actual carrying into them of a filling material which would obliterate them.

If this line of reasoning is correct, then the use of resin dissolved in chloroform can,

at best, serve only to paint very thinly the walls of the canaliculi and tubuli with a coating of resin. If this were impenetrable, as Dr. Johnston supposes, something might thereby be accomplished; but, as I have demonstrated and frequently published, such a coating of resin offers no resistance either to the passage of bacteria or to diffusible liquids. The experiments upon which these statements are based were made with bowspring resin, which was recommended by Callahan. Dr. Johnston said he used another kind of resin, but I believe all resins are soluble. I believe that all resins are freely oxidizable; and, if so, cannot be considered inert masses.

Dr. Johnston said tonight that resin fails in one point as an ideal root canal filling—because it is not radiopaque. This reminds me of an incident pertaining to dear old Dr. Callahan. A dentist conceived the idea of combining some barium salts with his chloro-resin, so he could radiograph it and show where it had gone. After he had made a number of pretty fillings, he took them to Dr. Callahan and said: "See what I have done with your chloro-resin." Dr. Callahan said: "How did you do this, and why?" The dentist told him, and Dr. Callahan said: "Remember this: we do not fill canals for the x-ray man, we fill them for the patient; and it is not so important that the filling should show in the radiograph as that the tooth should remain in a healthy condition."

Very little seems to be gained by the introduction of chloro-percha into the canaliculi. Radiograms of such results are interesting, but there is no stretch of the imagination by which we can conceive that these openings are perfectly filled after the chloroform has been dissipated. Our problem is not so much concerned with the perfect filling of spaces which it is obviously a physical impossibility to obliterate; but in the absolute destruction of or prevention of infection, and the avoidance of additional irritation by the agency of instrumentation, medication, or filling material.

Anyone who has a collection of radiograms can show dozens of cases of incomplete root-canal fillings, with a history of years of service and no evidence of pathology in pericementum, in lamina dura, or in bone. The frequency with which such results are noted leads to the belief that the method of filling the canal is not of the vital importance we have been led to believe. The misdirected energy, which many men apply to the attempt to accomplish the physically impossible in root-canal filling, might give better results if applied to efforts to sterilize the tooth and its environs, without adding insult to injury by chemical or mechanical trauma.

Last night they said they filled infected teeth as soon as they could get the root canals open, and used the Churchill-solution treatment, because they felt infection might recur if this was not done. Tonight they say that, in cases of recent devitalization, where there is no infection, they let the canals go for eight days, so that organic matter in the canaliculi and tubuli can decompose.[2] I hope Dr. Johnston will explain why he is not just as fearful of occurrence of infection during the interval while this decomposition is taking place.

If we would quit insulting nature by trying to cure something that is not sick and by trying to heal something that is not diseased, in cases of recent extirpation—if we would try to do our work without traumatism of any kind in the apical region, and fill these canals a little short of the end at the same sitting, and let the patient go—we would save a great many more teeth of this type than we ever will by any kind of medication, or by trying to force artificial things into places that nature can take care of very well.

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[2] The three papers under discussion were read on the evenings of November 7 and 8, 1921. The discussion was conducted on the second evening.

What has been said has not in any sense been a criticism of the work of the essayists, for they have submitted evidence that places their work far beyond disparagement. These points have been brought forward in the hope that they may be helpful to them and to you, in placing their work upon a surer scientific basis.

Dr. Rodriguez Ottolengui (New York City): As Dr. Crane said, I do not imagine I was invited here to tell Dr. Hinman and Dr. Johnston how much I love them, because I have told them that in Atlanta, Milwaukee, and in many other cities. I shall disagree with the essayists on certain points. I do so with considerable hesitation because I dislike to see a discussor show what he has done himself; and if I do this to some extent tonight, it is in a quasi-tentative way, and in the hope that I may give these gentlemen a suggestion or two that will lead their minds along other tracks and to a better solution than any I have found myself.

I fully agree with and commend Dr. Hinman for saying that many pulpless teeth, even when diseased, can be saved. He says: "I do not care how many other factors seemingly favor its retention—any destruction of this tissue (the apical cementum) condemns the tooth." I was rather surprised to hear that, because I have always looked

upon Dr. Hinman as prominently a surgeon in his work, and many of us are old-fashioned enough to believe in the operation of apicoectomy.

Dr. Hinman says that the radiogram alone is not sufficient for diagnosis. Later, he says that when a tooth is imbedded in a radicular cyst, it is not favorable for treatment. He also says: "The x-ray picture itself also indicates the presence of a cyst when there is a well-defined, dense, bony wall around a region of considerable destruction at the apex of the tooth." Also, that he can discover a flexibility of the overlying bone over the root on palpation. Unless I entirely misunderstand him, I would like to stress the point that you cannot determine the difference between a granuloma and a cyst by a radiogram.

Dr. Hinman says: "Therefore, the whole problem seems to me to be a question of sterilization of the sub-apical tissue in such a manner as not to lower its vitality nor destroy its function." It seems to me all of the gentlemen tonight have entirely overestimated the idea of sterilizing the apical region. Personally I do not think it is possible to sterilize any enclosed cavity in the body. I will speak of that a little more definitely when I come to Dr. Johnston's paper. I will show lantern slides of one case from practice. I brought these to show to Dr. Hinman, but now I am showing them to Dr. Crane also.

Formerly you would have said that wherever any extensive part of the apex of a tooth enters the diseased region or granuloma, as it does here (*fig. A*), it would be hopeless to try to cure it, except by apicoectomy, and we even went so far as to say that the root ought to be cut down to the line of living bone.

Fig. B shows the appearance of the tooth a month after the former picture was taken (*fig. A*), having been treated by electrolytic medication three times, a week apart, and then filled. You see clearly the extent to which the apex enters the destroyed-bone area.

Fig. C shows the tooth eleven months later. There is an amount of filling-in which surprised me. Let me say here that we can devise and advise all we please, but one cannot persuade a singer, as this patient was, to part with a central incisor. As old Dr. Atkinson would have said: "God and the angels saved her tooth," not Ottolengui and ionization.

Dr. Johnston spoke about "giving increased resistance to bacterial growth, etc." I want to point out that however valuable laboratory experiments may be, we are very likely to place an exaggerated value upon them. Last night the Doctor showed us a number of laboratory reports from which he concluded that he could sterilize a tooth and all the granuloma. He showed us how the tooth had been tested, what bacteria were present, and how they were ionized in the laboratory, or electrolyzed, after which he claimed they were sterile. In the first place the laboratory reports shown on the screen did not say that. The only deduction you could make from those reports is that the macerated teeth and soft tissues, planted on dextrose agar, failed to show a growth in forty-eight hours. When we were making similar experiments, we sometimes found growth after fifty hours; sometimes there was no growth on one, but growth on another.

Dr. Johnston claimed that because he could sterilize tissue, or tooth bone, in the laboratory, he could do so in the mouth; but that is not a logical conclusion.

Dr. M. L. Rhein (New York City): There are bound to be great differences of opinion, at present, in regard to technique. The many details are bound to be more or less debatable, for years to come. This evening's evidence of the salvability of teeth with devitalized pulps shows that we have developed within the profession, during the past decade, a group of men who have realized the possibility of saving a certain percentage of such teeth.

I have been very much disturbed by what has been written, particularly in the middle West and on the Pacific Coast, in opposition to this possibility. This subject has been considered in a very excellent manner by Dr. Hinman, and seconded by the man who followed him. I feel that I can safely say that here, in New York City, a very large percentage of the men who practise dentistry are satisfactorily doing this thing, in contradiction of the dictum of so many men in the West. I believe it means a great deal for the scientific future of dentistry.

Shortly after a meeting of the New York State Dental Society at Saratoga, a few years ago, I received a very lengthy letter from a member of the Novitzky School in San Francisco, congratulating me on the fact that I had "abandoned the effort to save dead teeth" (that is how the letter ran), containing a great amount of flattery, and welcoming me within their ranks. I responded to that letter to the effect that I supposed they meant by "dead" teeth, teeth in which the pulp had been devitalized; but that, as far as we understood the histology of the pericemental structures, in the East, we could under no circumstances accept the dictum that such teeth were necessarily "dead" teeth. Furthermore, I explained that he had entirely misunderstood my discussion of this subject at Saratoga. I stated that there was always a percentage of such teeth that could not be kept in a safe condition in the mouth. When that fact is well established, such teeth should be extracted as soon as possible, because they are a menace to the health of the individual. How serious that menace may be, can never be told from day to day. I received an answer to that letter that was just as defamatory in its nature as the other had been adulatory.

The saving of teeth in which the pulp has been devitalized is not an easy proposition. The longer I work in this field—and it has been my main work for forty-two years—the more I am convinced that success is attainable only with infinite patience, with attention to the most microscopic details, and with recognition of the necessity for attainment of a particular goal. There is no such thing in this particular operation as comparative success. Unless you reach a positive goal, the operation is a failure. In other words, we do many things in the practice of dentistry in which comparisons can be made—this operation is a little better than the other man's—but such comparisons do not hold in this very difficult operation. For that reason I have long since come to the conclusion that, when Dr. Ottolengui some years ago began an effort to place this operation in one of the fields of specialization in dentistry, he was promoting the proper idea. This is the only practical solution of the problem of treating pulpless teeth.

When the time comes that the practice of this particular branch is recognized as a distinct specialty (it should be ranked as much of a specialty as orthodontia), the general practitioner who is temperamentally unfit, or who finds it economically unprofitable, will not undertake it. Under the force of circumstances, many men feel that treatment of pulpless teeth belongs to the general practitioner, and that he cannot evade it without an admission of incompetency. Therefore, what happens? The community suffers from such a condition of affairs. There was a time, forty years ago, when I practised orthodontia, but I would not think of practising it today. When the time comes that that idea prevails in regard to this particular field of dentistry, the community is going to be better served. When the dentist feels he can safely say to his patient, "this is out of my line, I will refer you to someone who will be able to handle it," then we will not suffer rom some of the unfortunate spectacles that the oral surgeon is so fond of speaking about, and the physician so prone to criticise.

It is difficult for me adequately to express my feeling of gratitude to Drs. Hinman and Johnston for what they have given to us. While I differ with Dr. Johnston in much that he has said, I believe we are all working in the right direction. I am certain that Dr. Johnston, six years from now, will have very different ideas on some details from those he expressed tonight, because it takes a great deal of time to educate one's self sufficiently in this field—and then one realizes how little he knows.

I want to say a word about the matter of cemental injury that was discussed in this room by Dr. Arthur D. Black some years ago. The statement has been made, very often, that a tooth with exposed cementum might as well be extracted. The criticism I have to make of that statement, and various similar statements made tonight, is that it is impossible, in my opinion, to speak dogmatically of any disease as incurable. Dr. Ottolengui showed us a picture of a central incisor that illustrates the point that until we succeed, we never know what we cannot cure. The majority of such cases are incurable. But, what was the reason Dr. Ottolengui obtained such a splendid result in the case of the central incisor he showed us?

Those of you who heard my talk on this subject, here, last April (1921),[3] remember how strongly I dwelt on the point that electrolytic medication, unless carried to its logical limit, was nothing more than a toy. I dwelt on the fact that absolute sterilization was an impossibility and unnecessary—that what we seek to obtain bacteriologically, by our bacteriologic checking up, is an effective diminution in the number of streptococci. We do not get anything else in cases of true granuloma. There may be different conditions in the South that I am not acquainted with, but the gradual diminution in

[3] Rhein: *Journal of Dental Research*, 1922, iv, p. 327.

number of these bacteria is an indication that electrolytic medication eradicated the pathological condition. This is in accord with what Dr. Ottolengui has said in another relation and which he has repeated tonight. It is the most plausible theory. It is more consistent with our idea of the pathology of those parts than any other explanation of how electrolytic medication eradicates periapical disease, for it certainly does accomplish this end.

I showed on the screen, here, last April (1921),[4] a lower bicuspid that received something like twelve milliampere hours of electrolytic medication, largely with iodine, but the final one was with zinc. I undertook the treatment of that tooth under serious objection by myself. I told the patient that I thought it was impossible to save the tooth, but I was asked to use every possible means at my command to do so. To my own surprise, success crowned our efforts; you will recall the ideal encapsulation of the end of the root and the regeneration of the alveolar tissue. This result suggests that one should never say that something is incurable.

Dr. Johnston very insistently speaks against forcing gutta-percha through the foramen of the root. In the last picture Dr. Ottolengui showed on the screen, we saw that a large part of the root was absorbed; later, after root filling, all this destroyed root was restored by gutta-percha, some of which had penetrated the bone. The alveolar structure in which this gutta-percha was encysted had as normal an appearance, histologically considered, as I want to see. I know of no substance that is so compatible with the human dental tissues as base-plate gutta-percha. It is absolutely inert, it is non-irritating, and for that reason its protrusion has nothing to do with success or failure. Ten years ago my gutta-percha fillings protruded largely because I had not perfected the technique of working gutta-percha as it should be handled. Ninety per cent of the fillings I put in today have not as much protrusion as the fillings Dr. Johnston showed; and yet they give ideal encapsulation for the reason that I have been able to perfect a technique for the manipulation of base-plate gutta-percha macerated in chloroform. Dr. Johnston's failures in such cases were never due to the "button" of gutta-percha, unless that gutta-percha was infected when it passed through the foramen; which I doubt very much, knowing the methods of Drs. Hinman and Johnston. My own theory of the cause of failure, in the similar cases Dr. Johnston showed, is that it was due to insufficient amounts of electrolytic medication. I showed some failures last April that I believe were due to that fact. We must continue to apply electrolytic medication until we get satisfactory bacteriologic results. Dr. Johnston's bacteriologic work was criticised, I think very justly, because it depended on 48-hour negative reports. My bacteriologic tests were made at Columbia, and no report was sent to me before the conclusion of seven days of incubation. In many cases the first sign of growth appeared as late as the ninth day. If that is possible, of what value is a 48-hour observation?

On the question of resin, let me suggest that there may be a difference between "fiddle" resin and "window-glass" resin. My experiments have been made with the purest violin resin that I could obtain, as recommended by Dr. Callahan. The members of the Pathodontia Section remember the experiments we made with resin. It is a well-known fact, and it was demonstrated before that Section, that all resin masses contain organisms, and that when resins are placed in water or dilute salt solution, these organisms begin to appear inside of forty-eight hours. This was found to be true whether they were mixed with gutta-percha and chloroform, or simply with chloroform. We used a half-dozen controls in these experiments. When we used base-plate gutta-percha and

[4] Rhein: *Journal of Dental Research*, 1922, iv, p. 352.

chloroform in some of the same supply of distilled water and dilute salt solution, we found, even after two weeks, no change in the water. It was absolutely clear and free from organisms. Dr. Crane spoke of this in his discussion when he said that material in which resin was present was not inert, and is therefore absolutely incompatible with the effect we are after. We finally demonstrated that base-plate gutta-percha, chloroform, and resin, are incompatible. They can be mixed mechanically, but when the chloroform evaporates and the substance becomes hard, the gutta-percha has lost its flexibility. It is as brittle as glass; and, instead of being homogeneous in texture, is filled with holes like a piece of Swiss-cheese. This brittleness is a very bad quality, if there is any protruding gutta-percha, as it is only reasonable to suppose that, in the natural movement of the tooth in its socket, a brittle substance like this can be very easily fractured from the main position of the root filling; whereas, if it retained the flexible quality of base-plate gutta-percha, this would be very much less likely to occur.

In observing the various root fillings that Dr. Johnston placed upon the screen, there was distinct evidence of lack of homogeneity due entirely to the swiss-cheese character of the material. When base-plate gutta-percha is packed in a root canal, in combination with no other substance but the chloroform that softens and dissolves it, we avoid all direct pressure on this creamy base-plate gutta-percha. Our pluggers wrapped in cotton, which in turn is soaked in chloroform, are passed into the root canal. The canal takes the place of a mortar and the plugger that of a pestle. Our advanced technique consists in simply rotating the plugger (pestle) in the canal (mortar), which does not force the filling material in any particular direction. But by "mortarizing" it in this way, it is driven in every possible direction; and in this manner the filling material not only comes through the foraminal exits, but is forced into every crevice and space wherefrom organic material has been removed. The roentgenogram of such a filling shows an absolutely homogeneous mass, such as I have frequently exhibited on the screen.[5] It may be that, for the time being, they do not appreciate this difference of homogeneity, but I am convinced, from the splendid work Dr. Johnston has given us this evening, that it will be but a short time before he accepts this view of the situation.

I do not wish to leave the impression that, because of my many years of research in this direction, I am convinced that I *must* be correct in my technique, and that anyone who differs with me *must* be wrong. This accusation has been made so frequently that I earnestly resent it. It is most unjust to my deepest feelings on this important subject. I want to say in all seriousness that I am absolutely devoid of any personal ambition to have it appear that my technique is superlative. I say this because I am anything but satisfied with my own technique. It happens to be better than any other that has ever been shown to me; consequently, it is impossible for me truthfully to take any other position. Nothing would give me greater pleasure than to be able to say to Dr. Crane and to Dr. Ottolengui that I agree with them in the conclusion that the root filling does not necessarily have to insulate the root from the living tissue. I would love to accept the mixture of chloroform, gutta-percha, and resin, if I could consistently do so.

The fact that each of us is able to save a certain percentage of pulpless teeth means a great deal. I feel that we are joined together in a common cause to save, in their natural condition, as many human teeth as possible without detriment to the health of the individual. Our differences are purely technical. I trust that we shall be able to continue to discuss them, and differ with each other, without causing any impairment of the feeling of fellowship that is engendered by our having enlisted under a common banner to fight

[5] Rhein: *Journal of Dental Research*, 1922, iv, p. 341.

a great menace—the demand of many dentists that teeth with non-vital pulps should not be left in the human mouth.

Dr. Johnston (in conclusion): I am very glad that Dr. Crane was persuaded to come to New York to discuss my papers. I have read many of his writings, and have always learned something from each, just as I have learned tonight from his discussion of my papers. My main criticism is, in reply, that he does not differentiate strongly enough between the theoretical and the clinical. It is true that we need all the theory we can absorb, but we need it only to enable us better to understand and more correctly to interpret the practical or clinical.

As the discussion has been quite lengthy, I shall be brief in my reply. I may entirely omit some of the points that were brought out, to which I would like to reply, but which appear to have little bearing on the subject.

Dr. Crane's statement of the chemics and electro-chemics involved in this process is a most valuable complement to our papers and I welcome it. I could add little, if anything, to this phase of his discussion, neither could I or would I detract from it.

I fully agree with Dr. Crane that the term "ionization" is a poor one; "electrolytic medication" is much better. It is quite a "mouth-full," however, to say "electrolytically medicate a tooth." As the term "ionize" is universally used, and just as universally understood in this sense, I still use it. We use very many terms in an incorrect sense; yet their universal use, as stated by Webster, renders them correct. I fully agree with Dr. Crane that "a tooth cannot be ionized," namely, reduced to ions, but neither can we "fill" a tooth—we can only fill *cavities* in it.

My interjection of remarks on the purple or lavender color of electrolyzed iodine was simply to answer a question that is sure to come into the minds of many who may use this method of treatment. Of course, we all know that there are many things that will cause iodine to change its color, but I am sure that not many of us knew that the electric current was one of these agencies. I wondered about this change of color for a long time. After investigating, I found that iodine always assumes this lavender to purple tint when it is completely ionized.

When there is a marked difference in the caliber of the foramina of a multi-rooted tooth, the canals should, of course, be treated separately; or additional resistance should be obtained in the large canal by the partial withdrawal of the electrode, until the resistance of the large canal balances that of the small. However, where the foramina are practically the same size, though it may be theoretically advisable, I find it clinically unnecessary to treat them separately. This has been verified by bacteriological investigation.

In the first part of his discussion of fillings, Dr. Crane seems to deny that there is such a thing as a supersaturated solution, or that one liquid can be entirely replaced with another by diffusion, or that one liquid can be entirely replaced by a solid by the same method. That may be theoretically impossible, I do not know; but many things that are theoretically impossible happen every day. I have filled many minute glass tubes with chloro-resin and then worked in gutta-percha until a practical solid resulted. This is exactly what we do in the pulp canal.

I am not contending (in fact, stated exactly the reverse) that this method of filling is perfect—it is very imperfect—but I do say that I will gladly compare results, radiograms, or decalcified specimens, with any one. The man who can fill more solidly and imperviously the trunk canals, or more thickly "paint the walls" of the canaliculi, is the man I am looking for, and I will gladly discard my method for his; but Dr. Crane does

not mention such a method. It will be remembered that I stated that the ideal filling material must be incapable of shrinkage. We are very far from that now; but, with the other requirements in mind, it seems to me that chloro-resin-percha more nearly meets our needs in that regard than any other material of which we have knowledge, because of the fact that, properly placed, the shrinkage takes place from the center instead of the periphery and maintains its adhesion to the canal walls.

As for the matter of radiopacity of the pulp-canal filling-material: I admit that radiopacity is not necessary from a "filling" standpoint—it is necessary for the dentist, however. We dentists have a way of making a diagnosis without obtaining the history of the suspected tooth. It is seldom that we write for, or are asked for, the history of a tooth we are treating or have treated. If our fillings could not be shown with the x-ray, thousands of dentists would immediately say that given teeth were not filled; and then would either extract the teeth or enter them, drag out the fillings, and place others. That is why I say a pulp-canal filling must be capable of being shown by the radiogram. Another reason is that we could not demonstrate to our own satisfaction or any one's else that a canal was properly filled.

It is true, also, that we have reason to suspect the density of our fillings, in the minute canaliculi, but I maintain that it is much better to fill them as well as we can than to make no effort to fill them at all. The "stand-patter" has never been guilty of materially assisting progress in any line.

In regard to the matter of waiting eight days after the removal of a pulp before filling the canals: we feel justified in assuming that the tooth will not become infected because, the method of opening the tooth and removing the pulp being as nearly absolutely antiseptic as is humanly possible (not having carried any infection into the tooth), the chances of infection from the apical end are so slight as to be practically negligible. It is physically impossible to seal a wet apex with any of the accepted filling materials. It is physically impossible, also, to prevent exudation of serum in the apical portion of the canal from the freshly broken apical vessels.

Replying to Dr. Rhein's very excellent discussion and remarks: I am very sure that his prophecy that I will have "very different ideas in reference to some details" six years from now will be fulfilled. I believe that I will have different ideas a year from now, or less. Change is essential to progress, and he who does not change does not progress. Therefore, I hope my ideas may be changed through investigation and research continuously throughout all my years of practice.

The results of my investigations do not permit me to agree with Dr. Rhein that it is necessary to give the tooth as much current during the process of electrolytic medication as it will stand. I believe that the amount of current needed is a constant factor, which is not in any way controlled by the "endurance" of the patient. The amount of current I advocate, and the length of time it is permitted to flow, have been arrived at from both clinical and bacteriological evidence. I agree that *absolute* sterilization is unnecessary; but, instead of a gradual diminution in the number of bacteria, as Dr. Rhein states, I find that we can effect very rapid diminution—so rapid that, within thirty minutes, the field will be rendered practically and clinically sterile, if not actually so. I have not succeeded in getting a bacterial growth at all (5 to 10 days) when it did not take place within 72 hours.

The personal cases and those of Dr. Ottolengui, to which Dr. Rhein refers, are most interesting and enlightening, and to my mind show that the controlling factors in what can or cannot be remedied are the resistances and recuperative ability of the patient.

My principal objection to encapsulation, or other protrusion of gutta-percha, is founded on my inability to see the necessity for it. To my mind, one of the cardinal principles of pulp-canal therapy is that whatever is not actually indicated is contra-indicated. It is true that gutta-percha is a non-irritant but the chloroform that must accompany it, to permit its manipulation, is a violent irritant. If the apex is partly resorbed, I can see that a purpose might be served by encapsulation, but my technique calls for the extraction of such teeth. If there has been simply a loss of attachment in the apical region without resorption, as is stated by Noyes, the fibers of the periodontal ligament are still attached to the cementum. I think the slides shown here tonight prove that alveolar regeneration and reattachment of these fibers to the lamina dura will follow proper treatment.

While most of my bacteriological reports are based on 48-hour cultures, a great many of the cultures are incubated 72 hours or even longer, with identical results. I cannot see that it is clinically necessary to culture for the length of time Dr. Rhein advocates. If a bacterium cannot show growth within 48 to 72 hours, that bacterium is *clinically* dead. If it requires from seven to ten days of the most careful and scientific nursing and feeding to bring it back to life, it would not have had the faintest chance to survive had it been subjected, during this time, to the bactericidal forces of the human body.

My criticism of Dr. Rhein's method of using chloro-resin is that, either by starting with it too thickly mixed or allowing excessive evaporation, his finished experiments show the presence of a large excess of resin. These things must be used in their proper proportions and we must have the same proportion of resin to finish with that we had when we started. Otherwise, we cannot expect proper results. In regard to the presence of organisms in the resin: I must say that Dr. Rhein is the only one I know of who has found that he could grow bacteria which had been for any length of time immersed in chloroform, which all authorities agree is a powerful germicide. His bacteriologist evidently had some super-method for nursing these organisms back to life; and I say again that the human body would not afford them such careful nursing. Bacteria have been grown in Dakin's solution, but Dakin's solution is, nevertheless, an excellent germicide.

I have had the opportunity of watching Dr. Rhein operate and gladly accord to him the most perfect technique it has ever been my pleasure to see in the placement of gutta-percha in the canals. In my opinion his pulp-canal fillings are as nearly perfect as it is possible to obtain with the materials now at our disposal; but, as I stated at the beginning of my reply to his discussion, change is the essence of progress. I believe that Dr. Rhein has attained a maximum efficiency with his method. I am impelled to try other methods in the hope that I may discover something of benefit to the profession and public or, at least, dig out one or more truths which might possibly be of value to other investigators in the perfection of our ultimate pulp-canal technique. In the meantime, clinical evidence (which is the only conclusive evidence) indicates to my mind that my technique is at least as good as any other.

May I add that I appreciate greatly the opportunity of appearing before so many men who are so greatly distinguished in our profession. I thank you most sincerely for your great courtesy and hospitality, which I find to be fully equal to that of our own beloved Southland. [See the addendum, by Hinman, on page (59) of the introductory section for the volume, in this issue.]

[Illustrations of the papers by Drs. Hinman and Johnston, with the corresponding legends, which were not presented in time for publication with their papers (see foot-note 1), are presented herewith.—(W. J. G.).]

ILLUSTRATIONS OF THE PAPERS IN THIS VOLUME, BY HINMAN AND JOHNSTON, REFERRED TO IN THE FOREGOING DISCUSSION*

Fig. 1.—Large regions of infection on each root before treatment (see *fig. 2*). *Fig. 2.* Practically complete repair (see *fig. 1*).

Fig. 3.—A lower bicuspid, extensively involved (see *fig. 4*). *Fig. 4.*—Complete repair. Note absence of apical foramen (see *fig. 3*).

Fig. 5.—A small region of infection on an upper cuspid. Evidently the cause of a slight arthritis (see *fig. 6*). *Fig. 6.*—Complete repair. Arthritis disappeared two weeks after treatment; soreness to apical pressure disappeared. Patient and tooth perfectly normal four years later (see *fig. 5*).

Fig. 7.—Extensive destruction originating from a lateral foramen (see *fig. 8*). *Fig. 8.*—Apparently complete repair two years later (see *fig. 7*).

Fig. 9.—Extensive rarefying osteitis on an upper lateral (see *fig. 10*). *Fig. 10.*—Complete repair (see *fig. 9*).

Fig. 11.—A very large region of infection involving both roots and alveolar septum of a lower molar in a 10-year old girl. There was severe arthritis, with large swellings of both knees (see *fig. 12*). *Fig. 12.*—Complete repair in three months. The knees had returned to normal and all pathological symptoms disappeared in the same period (see *fig. 11*).

Fig. 13.—A typical small region of infection on an upper lateral (see *fig. 14*). *Fig. 14.*—Complete repair (see *fig. 13*).

Fig. 15.—Extensive condensing osteitis on an upper lateral (see *fig. 16*). *Fig. 16.*—Complete repair (see *fig. 15*).

Fig. 17.—Extensive involvement of an upper lateral. There was complete destruction of the apical half of the alveolar septum between the central and lateral (see *fig. 18*). *Fig. 18.*—Complete restoration of septum and lamina dura. The small "area" on the central apex evidently represents dense fibrous connective tissue (see *fig. 17*).

Fig. 19.—A lower central with two canals, both perfectly filled, though the presence of a second canal was not suspected.

Fig. 20.—The apical portion of a filling is too fine to show on the radiogram, but a minute "button" proves that the canal is perfectly filled.

Fig. 21.—An upper central with one large and one small apical foramen, both equally well filled by the "diffusion" method.

Fig. 22.—An upper molar with four canals, the fourth one being cut off from the pulp chamber by a deposit of secondary dentin. No instrument ever entered it. It was cleaned out and filled by diffusion through the communicating canal. An instance of an entire canal having been opened and perfectly filled without any knowledge of its existence.

Fig. 23.—An extracted tooth, filled and decalcified. It shows filling of a long communicating canal and of several "blind" canals. An extremely tortuous apical canal is perfectly filled.

Fig. 24.—A lower molar with mesial root filled and decalcified. It shows a remarkable diffusion of gutta-percha into the very delicate spaces connecting the two canals.

Fig. 25 shows no indication of infection. *Fig. 26* looks suspicious. *Fig. 27* condemns the tooth to the forceps. Each of these three radiograms was made from a different angle.

Fig. 28 indicates extreme resorption of the palatal root. *Fig. 29* is inconclusive. *Fig. 30* shows the palatal root undamaged, with a granuloma on the mesio-buccal root. Each exposure was made from a different angle.

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* Figures 25–30 illustrate Hinman's paper; *figs.* 1–24, Johnston's papers.—(W. J. G.)

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AMERICAN ACADEMY OF PERIODONTOLOGY

Proceedings of the Ninth Annual Meeting: Secretary's General Report, President's Address, and Reports of Committees[1]

ARTHUR H. MERRITT, Editor

New York City

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I. GENERAL REPORT BY THE SECRETARY

J. Herbert Hood

The ninth annual meeting of the American Academy of Periodontology was held at the Hotel Gibson, Cincinnati, Ohio, December 4, 5, and 6, 1922.

The president, Dr. Clyde M. Gearhart, of Washington, D. C., called the meeting to order at 2:00 p.m., on Monday, December 4. In the absence of the vice-president, Dr. J. O. McCall occupied the chair while the president's address was being read.

Dr. McCall introduced Dr. Robert H. Box, of Toronto, Canada, who read a paper written by his brother, Dr. Harold K. Box, on "histo-pathology of the periodontium," illustrated with lantern slides. The discussion of the paper was opened by Dr. Russell W. Bunting and Dr. Arthur H. Merritt, followed by Dr. Weston A. Price, Dr. J. Leon Williams and Dr. J. O. McCall. The discussion was closed by Dr. Robert Box.

The annual banquet was held at the Hotel Gibson, at 6:30 p.m., on December 4, and was attended by about one hundred and fifteen members and guests of the Academy. President Gearhart introduced Dr. C. H. Schott, of Cincinnati, as toastmaster, who had arranged a well appointed musical program by some members of the Academy and local artists.

Toastmaster Schott called on the following for after-dinner talks: Dr.

[1] Papers by Drs. Harold K. Box, and Richard N. DeNiord will appear independently in this JOURNAL as parts of the proceedings of this meeting. See page 435.

Weston A. Price, Dr. Olin Kirkland, Dr. Russell W. Bunting, Dr. Austin F. James, Dr. John G. Meisser.

The Tuesday morning session (December 5) included a report of the Committee on Fellowship and an extensive report of the Committee on Nomenclature and Scientific Investigation.

The Ohio State Dental Society, holding their fifty-seventh annual meeting in the Hotel Gibson, December 5, 6, and 7, invited the members of the Academy to be their guests at their sessions on Tuesday and Wednesday afternoons.

On Wednesday morning the Academy held a general session to hear the report of the Council and to elect the officers for 1923.

On Wednesday, at 8:00 p.m., the members of the Ohio State Dental Society were the guests of the Academy, at a symposium on focal infection, at which papers were read by Dr. Richard N. DeNiord of Buffalo, on "blood chemistry as a diagnostic aid in focal infection," and by Dr. John G. Meisser, of Rochester, Minn., on "focal infection in diseases of the urinary tract." These papers were discussed by Dr. Martin H. Fischer, of Cincinnati, Dr. J. O. McCall, of Buffalo, and Dr. Weston A. Price, of Cleveland.

The next annual meeting of the Academy will he held at the Hotel Statler, Cleveland, Ohio, September 7 and 8, 1923.

II. PRESIDENT'S ADDRESS

Clyde M. Gearhart

In 1914, I was invited to attend a meeting called for the purpose of organizing a National Society of Specialists in the field of Oral Prophylaxis and Periodontia. Since that time, it has been my pleasure to see this organization pass through a steady growth and become a recognized force in the dental profession. I have attended all the meetings, and have watched its progress with the pardonable pride of one who has had a part in its development. It is, therefore, with the keenest pleasure that I address you as president of this body.

Through these growing years, the members have worked unselfishly and in harmony for the upbuilding of the Academy. Many eminent members of our profession have encouraged us in the work we sought to do, and I take advantage of this opportunity to recount the splendid accomplishments of this organization in the advancement of dental science.

The American Academy of Periodontology was organized when there were but few who had the courage to announce themselves as specialists

in what we now know as periodontia. Oral prophylaxis as an agency for the prevention of caries was still on trial. The oral hygiene movement was just gathering momentum. Theories regarding the pathology and etiology of periodontal disease were empirical and necessarily discrepant, and the nomenclature of this branch of dental science was largely a mass of meaningless verbiage. Such papers as were presented on the subject dealt with it from two aspects; and the views presented by these two groups of writings had so little in common as to make it seem that two entirely different subjects were under discussion. There were, also, papers by scientists, or pseudo-scientists, who saw periodontoclasia only through the microscope or through the eyes of the medical pathologist. The writers of these papers had no clinical knowledge or skill. They took refuge behind a mass of borrowed pathological plumage and dressed their writings in a phraseology that carried conviction to the average dentist, who was overawed in spite of a feeling that everything was not just right. On the other hand, papers were presented by those who found a measure of success in the work of their hands and who dared to carry the message of success to their confreres. The latter group had been unable to make use of ideas of the pure scientists, but obtained results that convinced them they were in possession of at least a part of the truth. Unable to make headway individually, they nevertheless found that interchange of ideas strengthened their position. This was the state of affairs when the American Academy of Periodontology was called into being.

The first development, following the establishment of our organization, was the crystallization and unification of the ideas of treatment then held by those who were the most successful periodontists. It is worthy of note that much clinical success could be attained upon a slender basis of science. That there was truth underlying their work was evident.

The next stage in the development of the Academy was the analysis of the substructure upon which this success was built. It seemed often that pure science was disregarded; but gradually it was found, as is always the case, that what succeeded in practice was based upon scientific truth. Later researches, for instance the correlation of the work of Stillman and Box, together with the analytical work of McCall, proved the essential soundness of the hypothesis that had at first been propounded as a result of clinical observation. The Academy served as a medium of exchange for the ideas which were being developed by its members, and through this means the best thought was made available and the erroneous theories were discarded.

It was fortunate that, at this time, Box began his researches on the histology and pathology of the teeth and their supporting structures. A

periodontist himself, he was particularly fitted to weigh the evidence of the microscope with the evidence presented in the mouth, and strike a balance which should satisfy both the clinical and scientific findings. It is not too much to say that the Academy has, by the very reason for its existence, stimulated Box in his researches; and he has acknowledged the aid, on the clinical side, of its members. This fact, also, is a matter in which our members may take a just pride.

The work of Stillman, on traumatic occlusion, is too well known to need any comment. The very fact that this is so is, in itself, a tribute to the Academy; for without the means of disseminating and testing his theories, the value of his work in this field would have been acknowledged tardily by the profession, if at all.

The work of McCall, on nomenclature and etiology, is also well known. An outstanding achievement in dental literature is found in the volume, "clinical periodontia," by Dr. Paul R. Stillman and Dr. J. Oppie McCall. Nothing has been written on the subject that is so comprehensive as this text book. With becoming generosity they acknowledge indebtedness to a number of dentists and physicians as collaborators—among them, James, Merritt, Hayden, Spaulding, and our late lamented friend, A. H. Stevenson, all members of this Academy.

No confidence is violated when I say that Stillman and McCall confided to me that this Academy was their inspiration. McCall, like Box and Stillman in other fields, has acknowledged the aid he received, from other members of the Academy, in the development of his ideas; and he has stated that, without the Academy and its membership, his work in those fields would have been hampered and ineffective.

These names furnish instances in which the Academy has been a direct means of advancing the cause of periodontia. It is probably not too much to say that every member of the Academy has benefitted from its existence; and, moreover, the weight of opinion exerted by the Academy as a body has undeniably gone far to bring about acceptance of the views of its members by the profession at large. That this will bring lasting benefit to the public we serve is also without question. And so, when I say that the first nine years of the existence of this body are amply justified by its achievements, I know that I do not exaggerate but merely state a fact.

Shall we rest upon our laurels, that are achievements of the past? To do so would mean to lay ourselves open to criticism and to depreciation. This raises the question as to the path along which our energies may be best directed. A survey of the field of dentistry reveals an increasing activity in what is known as the oral hygiene movement. Practically

every country has awakened to the importance of mouth hygiene. Just as certain as the development of preventive medicine has marked a signal advancement and secured a permanent place in the profession of medicine, just so sure is oral hygiene taking a similar place in the field of dentistry. This movement has expressed itself in two ways. The first efforts of those who saw the possibilities of preventive dentistry were directed toward the education of the profession itself, and of the public, to the value of oral hygiene. The second expression of this group was the development of a procedure by which oral prophylactic treatment might be brought within reach of persons of moderate means, and particularly, the school children. In England, a strong impetus was given the movement, in 1920, by conditions among school children that were revealed by a parliamentary investigation committee. The government of New Zealand has appointed a national bureau of mouth hygiene, and several states in this country have taken similar steps. The achievements of workers in this field are common knowledge. Lectures, tooth-brush drills, motion pictures, statistical studies, and other educational means, have been invoked with a success that deserves our heartiest commendation.

In this country forward steps have been taken in dental hygiene by the training of specialists—by limiting the field to purely prophylactic work, to the cleaning and polishing of all exposed surfaces of the teeth. Recognizing the value of these prophylactic measures, and the special adaptation of women to this work, several states (about fifteen in 1922) have legalized the practice of dental prophylaxis by women. The development of schools for the training of women in prophylactic treatment has reached a point where it is evident that a new branch of the profession has been established. For several years much discussion has taken place as to whether the dental profession should recognize this new branch. Whether or not we approve of the ways and means by which the establishment of schools for the training of oral hygienists was brought about, oral prophylaxis and the oral hygienist are here to stay! It seems beyond question that those who are properly trained to give prophylactic treatment will be most helpful to those they serve.

In view of these facts, I believe that at least a part of the activity of this organization should be directed toward investigation of the standards prescribed for oral hygienists, to the end that they may become of even greater service to the public. The training of the members of this Academy should give them a particular interest in this field, and a special knowledge of the requirements. To no other body could this work be more properly entrusted. Will the Academy assume this responsibility?

There is little doubt that the members of the American Dental Association would look with favor upon a proposal for coöperation by this Academy in working out the destiny of the workers in this new field of preventive dentistry. And it is also probable that the graduates of the training schools already established would welcome the assistance this organization could render in the matter of raising standards. I earnestly recommend that a committee be appointed by the Council, to confer with the Oral Hygiene Committee of the American Dental Association and to report to the Academy. There are already signs of an outcropping of tendencies that may easily bring discredit on this promising branch of dental service. A movement should be inaugurated looking to the enactment of uniform laws in the states, that would establish the status of oral hygienists, and protect them from the encroachments and unfair competition of the untrained.

As some of you know, I have recently had an unusually long and enforced opportunity for deliberate reflection. A part of that time has been given to thoughts of the work this Academy could do. Our members have labored earnestly to establish periodontia upon a firm basis as a special branch of dentistry. We must not rest upon past achievements; we must go forward and broaden the field of our endeavors. I must admit that I have had strange misgivings from what I observed in the community in which I lived for several months; and have wondered whether, in other localities and other hospitals, the same improvident care of the oral cavity obtained.

I am jealous for the reputation of the Academy of Periodontology, and for our specialty in the dental profession, *periodontia*. I am particularly anxious that we assume that responsibility of leadership in all that pertains to the prevention of disease of the teeth and their investing tissues. In closing, let me quote to you from Article II of our constitution: "The objects of this Academy shall be to promote and advance the science and art of dentistry appertaining to the prevention of caries and the treatment of periodontal disease; to establish prophylactic and periodontic treatment as a specialty of dentistry; to provide for the dissemination through the dental profession of knowledge concerning these subjects; and to encourage the study of the relationship of these specialties to general medicine and hygiene." The thought herein presented I commend to your earnest consideration.

III. REPORT OF THE COMMITTEE ON FELLOWSHIP

Paul R. Stillman, Chairman of the Council; and Clyde M. Gearhart, Chairman of the Committee on Fellowship

The creation of Fellowship in this Academy was undertaken in response to the oft-recurring thought, among its members, that certain among them had made outstanding contributions to the science and art of periodontia, and that Academy membership alone seemed insufficient recognition of their service. In following this course, the Academy makes provision similar to that in other scientific organizations, which, in such wise, do honor to certain of their members. It is a fact, well known, that he who contributes the most invaluable service to mankind can seldom be repaid in things material. To such as these we can but extend our greetings as slight tribute.

One of our younger members has recently gained the distinction of being the first dentist to be awarded the Degree of Doctor of Philosophy, and that by a great University. This achievement is the more striking since the University of Toronto accepted his thesis on the preliminary qualification of the dental degree without insistence on the customary academic requirement. This act in itself is a tribute to the attainments of dental education. The honor is the more striking in that this thesis was upon a purely dental subject.

The research work of HAROLD KEITH BOX has already placed our profession under a debt which can never be repaid. In making him its First Fellow, the Academy has demonstrated that it has the capacity as well as the disposition to recognize distinguished service.

The consideration of further candidates for Fellowship has been based in large part upon the thought that the founding of this Academy constitutes an important service in periodontia. A review of the history of this organization indicates that the active work, which led to its founding, was carried on largely by Grace Rogers Spalding, Gillette Hayden, and John Oppie McCall. Were it only for the service thus performed, this body might well award Fellowship to these members. Each has, however, made important contributions to the upbuilding of our specialty and to its recognition as a specialty by the dental profession.

GRACE ROGERS SPALDING was among the early followers of D. D. Smith. It has been said, and undoubtedly it is true, that she has done more to demonstrate the soundness of the theory of oral prophylaxis and to secure its acceptance than any other of his adherents. Her ability in periodontic treatment has also been widely recognized. She is also the organizer of the

Oral Prophylaxis Section of the Detroit Clinic Club, an organization so well and favorably known as to need no eulogy. She has served as a member of the Council and as secretary-treasurer of the Academy, and has in fact been in almost continuously active work in this organization since its establishment. Her writings in this field have been of high quality and have served materially to advance the science of periodontology.

It was to be expected that the stock of Horace Hayden would bring forth another illustrious dentist. GILLETTE HAYDEN has proved that this expectation was well founded. Her Fellowship is recommended on solid personal achievement and without reference to her distinguished ancestry. Early after her entrance into the field of periodontia, her ability won recognition. Her brilliant mind, her incisive presentation, her just appraisal—all have combined to make her a valued contributor to the programs of many learned bodies. As a clinician she has demonstrated the practicability of periodontic treatment at a period when its success was questioned by many dentists. She has analyzed the work of other investigators, has sifted the evidence they have offered, and has proved with rare judgment wherein lay the truth. She has served on the Council of this Academy continuously since its organization, and was its second president. No member of the Academy has worked for it more steadily, more effectively, and with greater self sacrifice than has Gillette Hayden.

JOHN OPPIE MCCALL was one of the three who did the preliminary active work in the organization of the Academy of Periodontology. He served as chairman of the organization committee and drafted the constitution of the Academy. It was in this latter document that he first made an effort toward an improved nomenclature in this field. It is not too much to say that, at the present day, he is recognized as an authority on periodontal nomenclature. He was the first chairman of the Council and the third president of the Academy. He has served almost continuously on the Committee on Nomenclature and Scientific Investigation, and it was his work on this Committee which led him definitely into the study of the types and etiology of periodontal disease. He found the subject of periodontal etiology in a state of chaos. Theories on this subject were as numerous as were the individuals attempting to treat dental periclasia. He analyzed the situation, assigned to each theory its proper value, and correlated the available information. The result of this was to systematize the existing views in this science, and to present an orderly grouping of previously discrepant theories. His work on the etiology of periodontoclasia has yet to be challenged. This alone would make him worthy of the honor we propose. His ability in presenting the views of the advanced workers in

the field of periodontia has caused him to be in demand as an essayist in our societies. His contribution to the periodic literature and his skill as a clinician have served to advance the science of periodontology. He has, furthermore, demonstrated his ability as a teacher through years of continuous service upon university faculties. As a co-author of the Text-book of Clinical Periodontia, it is my pleasure to acknowledge the contribution which Dr. McCall made to that work. If this book is adjudged to have merit as a literary presentation, the credit for this must go to him. Through his mental attributes and by his training, he has made its contents acceptable. If others knew him as does the writer of this report, it would be evident to all that to no one more worthy could the distinguished honor of Fellowship of this Academy be awarded.

This report is submitted by the retiring Chairman of the Council, because of the fact that it was the sense of the Committee that one of its members should be recommended for Fellowship.

IV. REPORT OF THE COMMITTEE ON RESEARCH

Andrew J. McDonagh, Chairman

The results of research, in various parts of the world during the last ten years, show that conditions of the body are either controlled or greatly influenced by diet. This is true of all parts of the body, the periodontal tissues not excepted. Furthermore, during the formative period at least, the character not only of the periodontal but also of the dental tissues is governed by the food ingested.

The work of Dr. and Mrs. Mellanby seems to leave no doubt about the foregoing. The experiments of Mrs. Mellanby, on dogs, show that the form and quality of the bones of the jaw and the alignment of the teeth, depend to an almost incredible degree on the constituents of the food consumed during the formative period. Consequently, the early feeding of the growing animal carries its influence, so far as the periodontium is concerned, right through the span of life. But food is an important factor not only in the growing animal, but also in the adult as well. Scurvy, which is solely the result of deficiency in the diet, manifests itself as a destroyer of the periodontal tissues, resulting in many cases in one form of the disease we recognize as dental periclasia. Until recently the origin of this phase of dental periclasia was not recognized because we had not thought of scurvy in any sense except as we see it in an extreme case. We are indebted to Dr. Alfred F. Hess for the knowledge that one of the effects of a scorbutic condition is such a degree of deterioration of the walls of the blood vessels

that the blood breaks through and the gums bleed before other symptoms of scurvy are manifested. A prolongation or recurrence of this stage of scurvy may be more frequently a factor in the etiology of dental periclasia than is generally conceded.

Dr. Eugene Talbot, in his work on "interstitial gingivitis" (another name for dental periclasia), gives full credit to the assimilation of food and to metabolism. His work, if followed to a logical conclusion, will not be far from the facts deduced from the later study of conditions resulting from a scorbutic diet. But it was left to Dr. G. V. Black to prove, by his experiments, that the amount of food ingested by the individual was responsible for the amount and kind of salivary calculus deposited on the teeth of that individual. By repeated experiments he showed that, if a person takes and can digest so much food that the quantity of globulin and calcoglobulin produced exceeds the amount the tissues can immediately use or store, those excessive nutritive materials appear in the secretions. Consequently the saliva will be overloaded with them and, as a result, a deposit of salivary calculus lodges on the teeth. Vitamines, and the balance of the lime salt content of the food, undoubtedly have some influence in this connection, but experiments along this line are not sufficiently conclusive as yet to justify a definite statement.

Study of the influence that the endocrine glands exert over dental periclasia is just in its infancy, but apparently the action of these glands is influenced greatly by the constituents of the food; as an instance, the effect of vegetables containing iodine on the thyroid gland. No doubt the parathyroid and other ductless glands are similarly affected by various food ingredients, and we now treat some phases of periodontoclasia with these facts in view.

A year or so ago the members of the Academy of Periodontology received questionnaires which I sent them, also a request that they fill them out and return them to me. Some have complied, a few have not. I also sent these questionnaires to a number of dentists who are not members of the Academy, but were believed to be interested. In all about five hundred were returned. Many interesting facts are disclosed by the answers given. The information, however, is more negative than positive; in other words, it tells us that certain food habits, which some enthusiasts claim are the cause of dental and periodontal troubles, cannot be relied on to prove these theories. The diet ordinarily used on this continent by the adult seems to be adequate for nutrition; the appended summary of answers shows, however, that there is a possibility of injury to the periodontal tissues and probably to the dental tissues of the adult.

Summary of returns on the relation between diet and dental conditions

A good proportion of carbohydrates, and lack of raw vegetables and raw fruits
26 per cent have healthy periodontal tissues and no pockets
74 per cent have pockets or unhealthy periodontal tissues
45 per cent have numerous cavities or fillings
55 per cent have very few cavities or fillings

A good proportion of raw vegetables and raw fruit, but lack of carbohydrates
24 per cent have healthy periodontal tissues and no pockets
76 per cent have pockets or unhealthy periodontal tissues
42 per cent have numerous cavities or fillings
58 per cent have few cavities or fillings

A good proportion of carbohydrates, raw fruits, and raw vegetables
36 per cent have healthy periodontal tissues and no pockets
64 per cent have pockets or unhealthy periodontal tissues
52 per cent have numerous cavities or fillings
48 per cent have few cavities or fillings

A good proportion of carbohydrates with milk, but no raw vegetables or fruit
37 per cent have healthy periodontal tissues and no pockets
62 per cent have pockets or unhealthy periodontal tissues
56 per cent have numerous cavities or fillings
44 per cent have few cavities or fillings

A good proportion of raw vegetables and raw fruit with milk, but lack of carbohydrates
27 per cent have healthy periodontal tissues and no pockets
73 per cent have pockets with unhealthy periodontal tissues
36 per cent have numerous cavities or fillings
64 per cent have few cavities or fillings

A good proportion of raw vegetables and fruit, milk, and carbohydrates
35 per cent have healthy periodontal tissues and no pockets
65 per cent have pockets with unhealthy periodontal tissues
58 per cent have numerous cavities or fillings
42 per cent have few cavities or fillings

As far as these returns are concerned, we must remember that the dentists were asked to send reports of the first cases coming into their offices after receiving the questionnaires. Therefore, the majority of these are not selected cases but represent the usual run of patients—in other words, the average individual and the average diet. On the whole, the latter is probably fairly well balanced. A few exceptional cases were reported, which I will relate, as they bear on certain phases of the situation.

One outstanding fact seemed to be evident throughout, namely, the almost universal connection of worry with dental periclasia. Of all the reports received, less than 2 per cent of the number of patients, who had periodontal destruction in any advanced degree, were reported as *not* worrying. Furthermore, those comprising the 2 per cent follow avocations which would ordinarily cause a nervous strain, as for instance school

teaching and law. One dentist reported that two patients of his had stopped the habit of worrying after he treated the cases successfully; another reported the same thing about one of his cases. This would lead one to suspect that nervous conditions have some connection with dental periclasia.

One of the most interesting reports came from Dr. W. A. Fleming, who says a family living in his neighborhood acquired at the same time two dogs of the same age, one a mongrel, the other a pet house dog. The mongrel was put out of the house to live and fed as that sort of dog usually is, being allowed to forage in part for himself. The other, the house dog, was tenderly cared for and fed the same thoroughly cooked food as that served to the family. The mongrel kept his teeth, to old age, in good condition. The house dog lost practically all of his teeth, because they became loose and the investing tissues were destroyed. Was this result due to the breeding, the environment, the kind of food, the quantity of food, or what?

We all have many cases in our own practices which tell their lessons, but we fail sometimes to get all out of them we might. I will close this report by relating one I had to deal with—the case of a young man. The gingival tissues surrounding his upper anterior teeth, from cuspid to cuspid, were enlarged to twice their normal size, extended over the teeth to near the incisal edge, and were composed apparently of fairly dense fibrous tissue of a very dark color. There was a scant exudation from the gingival crevices, which, of course, were unusually deep. This exudation ceased, however, when the gingival crevices were thoroughly cleaned out. There was scarcely any bleeding even under pressure, or manipulation, or instrumentation. The color, to which I referred, was very dark blue, in some places almost black. This patient was a fruit grower and fruit salesman, and practically lived on apples, eating as many as two dozen large ones at a time, without taking any other kind of food. By changing his diet, and by local treatment, we were able to reduce the size of his anterior ula to nearly normal and to induce a better color. Was this due to endocrine disturbance, or to fruit acid affecting the lime salts of the blood, or to some other cause such as deficiency of proper food that the excess of fruit replaced?

This report, which is really one of progress, is very disappointing, I know, inasmuch as there is little of a conclusive nature in it; but, in conjunction with other work of this kind, it may be of some use. I sincerely hope it will; or rather, I sincerely hope some one will take up this work and do what, up to the present, I have failed to accomplish.[2]

[2] The Committee has had the assistance of Dr. Marjorie Milne, who tabulated the data and calculated the percentages for this report.

DENTAL EDUCATION[1]

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I. INTRODUCTION

The fact that dental education has reached the pinnacle toward which, for years, it has been climbing gives the initial impulse to this outline sketch of that long upward struggle. The Carnegie Survey of Dental Education marks that summit. It is an epochal event in dental education. Its consequences to dentistry are certain to parallel in beneficial results the sweeping consequences the Carnegie Survey of Medical Education had for medicine. For many earnest educators this reflection warms the cockles of the heart!

In writing this brief and hence inadequate sketch of the development of dentistry, I shall limit myself to the general trend of that development as it has affected modern dental education. This implies the omission of several highly interesting but self-limited systems, such as the old Etruscan, the pre-Incan and the Oriental systems, as well as any consideration of pre-historic methods. Personalities, too, however important, can receive but scant attention. I shall shut my eyes, further, to the tempting by-paths opening on every hand into the curious and often shocking fields of old-time dental practices, and keep to the high road—not always, alas, clear-cut—of scientific progress.

[1] Address at a meeting of the William Jarvie Society for Dental Research, of the School of Dentistry of Columbia University, at the School of Dentistry, October 26, 1922.

II. ANCIENT DENTAL HISTORY

At its inception dentistry stood, as it will stand at its maturity, in the relation of child to parent. (Today that child is the prodigal son.) As early as 1550 B.C., in the famous Eber Papyrus, we find record of a healing fraternity in Egypt, of which the dentist was fellow member with the internist and the oculist. At the time the Egyptians, you will remember, "possessed all knowledge," and had made medical knowledge practically international. Darius, the great Persian, kept Egyptians as his court physicians. He said of them: "Medicine is practised among them on a plan of separation; each physician treats a single disorder and no more; thus the country swarms with medical practitioners, some undertaking to cure diseases of the eye, others of the head, others again of the teeth, others of the intestines, and some of those which are not local." Egyptian mummies preserve for us artificial teeth, with evidences of mended fractures. There is no doubt that, in this most highly perfected of ancient civilizations, dentistry constituted an integral part of medical knowledge.

Eleven centuries later, at the time of Hippocrates, "Father of Medicine," oculists and dentists were specialists in medicine. Practitioners of that science were held in high respect and were men, often, of liberal education. Dentists took, with other specialists, the fine old Hippocratic oath, which placed them on honor to practise always "for the benefit of the patient," and "with purity and holiness." This fact is worthy of especial note, for even today Hippocrates is held in high esteem. He was "the great creator of scientific medicine and of artistic practice." His "undying importance rests not so much upon his enrichment of science with new material (though this honor too is his unquestioned due), as upon the creation of a scientific (lay) medicine and art; upon the method and really great principles which he introduced for all time into science and especially into practice." He "lives and will continue to live the immortal model of medical thought, and still more of medical practice, so long as medical science itself exists" (Baas, "History of medicine"). Osler, in his great book on the evolution of medicine, cites, as the outstanding contribution of Hippocrates and his school to modern medicine, the *art of careful observation.* For the first time medicine held a detached, scientific attitude—it "broke its leading strings to religion and philosophy." Many of his voluminous writings deal with the teeth, as a part of the bony system. That dentistry had an integral part in Hippocrates's scheme of thought is a consideration of some moment to us.

For the time of Aristotle we have no specific mention of dentistry. The

Alexandrians, however, operated on teeth as on any other portion of the anatomy. For Roman times, we learn, from Aulus Cornelius Celsus, an authoritative writer on medical subjects, the current method in treating diseases and injuries of the teeth. He describes "extraction of teeth by means of forceps, fastening teeth with gold wire, bursting hollow teeth by peppercorns pressed into them, etc. The results of extraction in cases of ankylosis of the teeth and alveoli, caries and necrosis, are known to him, and the tooth was accordingly shaken loose most painfully before application of the forceps."

These archaic methods provoke a smile today. I need not explain that I do not quote them as evidence of early professional attainments in our specialty. What I wish to emphasize is the fact that in this period the fundamental conception of a dentist was still that of a *physician*, hence, of a *learned man.*

After the Saracen invasion (640 A.D.) dentistry shared with surgery its curious divorce from medicine. An aristocratic doctrine prevailed among the Arabians which forbade a physician to do aught with his hands. His field was limited to giving advice as to food and medicine, any operation being left to his assistants! Dentistry, naturally, fell also to them, and was not practised by the higher class of physicians. Shortly before the overthrow of Saracen civilization by the Turks, in the 13th century, we received the writings of one Albucasis, whose work was designed to raise surgery to something of its ancient dignity. A large part of this work on general surgery is devoted to operations upon the teeth. (Extraction, in his time, was left almost entirely to the barbers.) His methods were somewhat in advance of his predecessors, and his writings on the subject of the teeth by far the most exhaustive recorded up to that time.

III. DENTISTRY DURING THE MIDDLE AGES AND THE PERIOD OF THE REVIVAL OF LEARNING

As is well known, medicine suffered a decline throughout the Middle Ages, descending at last to the monks and the itinerant Jews. Surgery fell into the hands of barbers, farriers, and swine doctors, who made common company in 1308 with the London Company of Surgeons. Fortunately, however, a struggling university had preserved here and there a stray germ of the old Hellenic culture. The Turkish capture of Constantinople, in 1450, drove crowds of learned Greeks into the European universities; and, with their coming, learning once more burst into flower. Spain, France, Italy, and England were the gainers. In their universities, medicine rose rapidly into its old position of importance.

To a group of Roman scholars, Vesali, Ingrassias, Fallopia, and Eustachi, surgery owes much of its rehabilitation. The last two in particular devoted much intelligent study to the teeth. For the first time a really intelligible and useful description of these organs was published. This paved the way for the publication, twenty years later, in 1581, of the first work explicitly on the structure and diseases of the teeth. Its author, Urban Heimand, was a general surgeon. Despite these learned names, however, surgery was long degraded by its association with the barbers, whom it was unable to shake off.

An historical sketch on dental surgery published by James Snell, dentist, in 1832, has an interesting paragraph on this situation. "The surgeons of Paris," he writes, "who since the time of Lanfranc, had constituted a particular college, had before the commencement of the 16th century, made reiterated complaints on this subject, to the faculty of medicine; the only result of which was, that they were allowed to enjoy a certain pre-eminence over the barbers, on paying sixteen sous annually to the said faculty. Things became worse, however, soon afterwards, the physicians of Paris having in 1505 concluded a treaty with barbers, by which the latter were, on certain conditions, admitted members of the faculty, and dignified with the title of Tonsores Chirurgici, instead of Barbitonsores, by which they had previously been known. Ten years afterwards, Barat, the President of the College of Surgeons, by addressing his complaints to the University of Paris, instead of the Faculty of Medicine, obtained for the surgeons a partial emancipation from that state of vassalage in which they had hitherto been held by the physicians; and the college was soon afterwards acknowledged as a learned institution, with the privelege of constituting its own Masters, Bachelors, Licentiates, and Doctors in Surgery. These privileges, in spite of the frequent and vexatious opposition of the Faculty of Medicine, the surgeons still continued to enjoy; so much so, that in 1596, they issued a decree, by which the barbers were compelled to call in a sworn surgeon, in every case of importance, and to confine themselves entirely to the most trivial accidents; but, as the majority of those which befall the teeth, are commonly considered in that light, it is probable that the barbers of this period were still the principal dentists of the age. We hear of an oculist about this time, as distinct from a general practitioner in surgery, in the person of Bartisch, oculist to the Elector of Saxony; but of a professed dentist, no mention is hitherto to be met with."

IV. DENTISTRY IN THE 17TH AND 18TH CENTURIES

Dentistry, it will be seen, had fallen on bad times. But, with the great advancement in science of the late 16th and the 17th centuries, a change was effected for the better. It is once more in the best practice, we must particularly note, *a field of medicine.* I quote again from Snell: "The advancement thus rapidly effected in natural philosophy soon extended itself to medicine; and the accumulation of new facts, in every branch of this extensive science, was, in a short time, so great, that a more especial attention to a few departments alone became indispensable, in order to excel. Hence arose, among medical men, a more minute division of labour than had been known since the time of the Egyptians; and this minute division of labour was, in its turn, a cause of still greater progress in the several departments of medicine, by concentrating the energies of individuals, to particular branches, instead of dividing them among the whole. It was then at this period, that the dental art began to be exclusively cultivated by a certain class of medical practitioners, and that we now, for the first time, meet with the appellation of surgeon-dentists, a title given, so early as 1622 to Gillies, and others, in France, but not for many years afterwards fully established. . . . . It was not till the year 1700 that persons destined for the profession of dentists were compelled in France to undergo a regular examination; and it is from this period, perhaps, that we must date the establishment, in modern times, of the dental art as a distinct branch of medical practice."

During the 18th century great progress was made in restorative dentistry, as well as in fundamental anatomical research. France continued to contribute men of note; for instance, Pierre Fouchard, author of the first complete work on dentistry (1728) and "the founder of modern scientific dentistry." That book, Philipp Pfaff's *Abhandling von der Zähnen* (1756), and John Hunter's "Natural history of the human teeth" (1771), form three important landmarks in the history of dentistry. Hunter was essentially "a great all-round biologist like Haller and Johannes Müller, and, with Paré and Lister, one of the three greatest surgeons of all time. . . . Hunter was the first to study the teeth in a scientific manner, and the first to recommend complete removal of the pulp in filling them. He introduced the classes, cuspids, bicuspids, molars, and incisors, enlarged upon dental malocclusion, and devised appliances for correcting the condition" (Garrison, "History of medicine.") . . . His "Practical treatise on the diseases of the teeth" initiated, in England, a new epoch for the dental art, which, abandoning its blind empiricism, began to take its stand on the basis of rigorous scientific observation" (Guerini, "History of dentistry.")

A notable 18th century dentist of the biological type was Robert Bunon, a Frenchman, whose works on dentistry have a high scientific importance.

Contemporaneously, as always, there were quacks in great numbers. Baas, writing of Germany in the 18th century, says: "the cosmetic art of dentistry, an art then, as to some extent at the present day, the field of charlatans, enjoyed much cultivation." He then enumerates six men who to his knowledge were *not* charlatans! In fact, Baas states that John Hunter "did not scorn *even* the subject of dentistry!" The significant point for us is that, however outnumbered by the quacks, there were still men of the surgeon-dentist type who held to the straight and narrow path of scientific study.

V. RISE OF MODERN DENTISTRY

The French Revolution, with its general lowering of barriers, played havoc with dentistry. "Short-cut" men sprang up throughout Europe. America, too, had its share of them. We have little early record, in fact, of the American scientific practitioner; we have many accounts of the skilled mechanician, notably of the erstwhile silversmith, Paul Revere. The first forty years of the 19th century in America were marked by the continued development of this type of skilled technician. The only training afforded, beyond the apprenticeship to a practitioner, were four- or five-month lecture courses in medical subjects, such as anatomy, physiology, and materia medica. Most dentists practised without medical training and without degrees. Naturally, the emphasis fell on technic. Dr. C. R. Turner (*Dental Cosmos*, January, 1920) points out: "As more was known about these matters than about what may be called the vital relationships of dental operations, and as mechanical skill was necessary to the actual practice of dentistry, it is not surprising that mechanical training should have taken so large a part in dental education. As this was the greater part of the course, it is natural that it should have become its mort important part, and that dentistry should come to be regarded very generally from a mechanical point of view. Furthermore, as the empirical method prevailed very largely even in medicine, it is not difficult to understand how dentistry, consisting as it did of methods of practice learned by experience, should have followed similar methods in its educational efforts. . . . . The impetus of this development of mechanical ideals underwent great elaboration from 1875 to 1890, for during this period crown- and bridge-work entered upon a period of rapid growth. The evolution of orthodontia during this period was also distinguished by the infinite variety of mechanical appliances which its votaries produced. This may be truly said to

have been the mechanical age in dental education, and the early empiricism was continued by the fascination of the study of mechanical arts."

This quotation carries us beyond the establishment, in 1839, of the first dental school. The University of Maryland had been asked by two prominent practitioners, Drs. Hayden and Harris of Baltimore, to establish a course in dentistry, and with lamentable lack of foresight had refused. Dissatisfied with existing conditions, and having failed in this undertaking, Hayden and Harris finally brought about the establishment of the Baltimore College of Dental Surgery.

This sharp cleavage has been called a "crime." Unfortunate in its consequences it certainly was; but it was a natural outgrowth of the chaotic scientific conditions of that time. So close a connection has this fact with the subsequent development of dental education that I must digress for a brief account of such conditions, especially in medicine.

VI. DEVELOPMENTS IN MEDICAL EDUCATION AND DENTAL EDUCATION COMPARED

Medical training then and later was haphazard. There was no such volume of medical knowledge as was opened in the latter half of the century, with the flowering of bacteriology and the other biological sciences. Medicine was a three-year course with no preliminary requirements except a high-school education, and that, in the private schools, was often blinked. These early schools, established first to meet a real educational need, were soon augmented by many others seeking only profit. In such schools clinical instructors often paid down a tidy sum for the privilege of holding chairs. This they expected to get back in the benefit to their private practices from such a teaching connection! It was often returned many times over, as the college grew, in student lecture fees and in referred cases. There was a dearth of trained scientific teachers and very little laboratory work. Often clinical men taught all the science offered. Such medical schools continued to spring up as late as 1900.

Meanwhile, however, there had been a growing sense, among practitioners trained in the university medical schools and among scientists, that the situation was intolerable. Huxley's ideal had been steadily permeating the best medical thought, which, as always, was found in the few university schools then contemporaneously developing. Their disinterested efforts finally led to a great house-cleaning, under the Carnegie Foundation, with Abram Flexner at the head of the survey. The publication of the Foundation's report spelled doom for the proprietary schools. Fifty per cent of them disappeared within a year. I need not enlarge on the sweeping results of

this published report to medical education. It is too well-known to require comment.

Dental education from 1839 took the same general trend. The early private schools, established to fill a real need, were rapidly added to, toward the close of the century, by schools set up purely for profit. The temptation to teach with an eye to the owners's pocketbooks rather than to the advancement of science grew overwhelming. With a continually growing demand for dentists the number of these schools increased, and the standards fell lower and lower.

Again the saving phase of an apparently hopeless situation lay with the universities. In 1869 Harvard took the step, momentous to the future of dentistry, of establishing a dental school. Three years later it graduated its first class, with Dr. Oliver Wendell Holmes as the speaker of the day. His words are pertinent to our discussion:

"This Commencement of the Dental School has a real significance, though it makes little show, and does not appeal to any vulgar interest. It publishes the fact that a new pursuit has been assigned its place among those chosen professions which a fully-organized educational institution may fitly take in hand, and provide for teaching. And you may be assured, that, before our old university would take such a step, its governing boards had satisfied themselves that the time was fully ripe for it. The dental profession had achieved its success, and had won its place in the estimate of the intelligent public, before its teachers were asked to share the labors and the dignities which belong to the faculties of this great institution. A few generalities are all that can be attempted in a discourse like this; enough to give some little idea of what the dental profession has grown out of, and what it has grown to; a few hints to make us feel more keenly its importance; a picture or two of old superstitions and fancies and barbarisms to contrast with the enlightened knowledge of our own time; a brief mention of some of the leading modern improvements in the scientific and practical departments relating to the teeth; an explanation of the causes which have kept the dental profession from receiving the recognition it has a right to claim; and a vindication of its title to the regard of the community, and to a fair representation of its teachings at the great seats of learning." Further on Holmes alludes to "the branch of the medical profession to which this graduating class has devoted itself." At the close of his address he states, "your profession, young gentlemen, is now an accepted province of this great and beneficent calling (medicine). . . . . Harvard University is doing all it can do to recognize the value of your profession to the community; and it does this at the time when it is making the most strenuous efforts

to place medical education on a basis worthy of a branch of knowledge so complex, so vast, so all important to mankind."

I quote this at length because it leaves no doubt as to the status of dentistry in his regard.

Other universities soon followed the lead of Harvard. Michigan established a dental school in 1875, Pennsylvania in 1878. In 1921 two-thirds of all the schools were parts of state or endowed universities. The list is still growing.

VII. RECENT PROGRESS IN DENTAL EDUCATION

In 1884 various schools of all types organized the National Association of Dental Faculties for the purpose of standardizing and regulating dental education. This body, sincere in its purpose, did a great deal toward bettering conditions. There was a great deal to be done. I trust I have not made it appear that at any time in its history dental education has moved steadfastly toward a clearly perceived goal. Quite the contrary. Like all other human institutions it has muddled along in very human fashion, with—as one looks back—an astounding lack of vision, even of foresight. As late as 1884 it was necessary for this Association to take very elementary steps toward betterment. At its first meeting it "abrogated the prevailing custom of allowing five years of practice to be substituted for one session of college work, recommended the general adoption of a graded curriculum of two years, recommended the subjects of instruction, and declared itself in favor of an examination upon the rudiments of an 'English education' for all candidates for admission. In 1889 a three-year course, of not less than five months each, was adopted to go into effect in 1891–92. Six-month sessions were approved in 1894, and extended to seven months in 1899–1900. In 1901 a four-year course was projected for 1904–05 to accommodate the rapidly increasing subjects, but in 1904 this was rescinded in favor of a three-session course of thirty weeks each, as it was decided that the adoption of a four-year course had been a little premature. The four-year course went into effect, in all the colleges of the country in 1917.

"The National Association of Dental Faculties advanced the preliminary educational requirement, in 1897, to admission to a high school; and, in 1900, to admission to the third year of high school. In 1907–8, three years of high school were required and now (1920), for several years past, a completed high-school course or its equivalent is necessary for admission to dental colleges. Thus will be seen the influence of this association in lengthening the dental course and in prescribing preliminary educational

standards" (Dr. Turner, *loc. cit.*). Once more "the mills of the gods grind slowly."

One by one most of the university schools withdrew from the National Association of Dental Faculties. Harvard was the first to go. In 1908 six of them formed a group with nominally the same ideal—the disinterested advancement of scientific dental education.

The first steps agreed on by this body, the University Faculties' Association of American Universities, were (1) the necessity for a survey of dental education, (2) the need for lengthening the course. Impetus was given to both movements by Dr. Hunter's momentous charges against American dentistry in 1911. One school initiated a four-year course in 1915; others followed in 1917. Since its organization the University Association has bent its energies toward raising entrance requirements, standardizing the course, lengthening the course—it is now five years in many universities—and otherwise establishing the claim of dentistry to rank as a learned profession. It has been vastly heartened by the encouragement of such men as Dr. C. H. Mayo, Dr. John B. Murphy, and several other leaders of scientific thought.

VIII. DENTISTRY A BRANCH OF MEDICINE

In 1913, Dr. Mayo, in a paper read before the Chicago Dental Society, stated that "the next great step in medical progress in the line of preventive medicine should be made by the dentists. The question is 'Will they do it'?" (*Dental Review*, April, 1913, p. 297). In 1918, Dr. Mayo, at the fiftieth anniversary meeting of the Dental Society of the State of New York, made the following comment: "Modern dentistry is relieving the world of much of its misery by watchful care of foci connected with the teeth, and the trend of modern medicine and dentistry is bringing their fields again closely together. Dentistry should be a department of medicine, as it is as closely associated with medicine as are the specialties of the eye, ear, nose and throat, etc." (*Dental Cosmos*, November, 1918, p. 965). In May, 1922, Dr. Mayo, in addressing the same society said flatly: "It is more important for the dentists to have a good foundation in medicine and anatomy than for the oculist to have it, because in the mouth will be found evidence of more diseases than in any other region of the body; such evidence will be in the form of nutritional changes, and infections around the gums, teeth, and in the tonsils. I believe the doctor, the veterinarian, and the dentist should all take the same course for the first two years of their college work and after that take separate courses in the study of their professions. There is no use of trying to better the practice of dentistry and have persons go into it

without any idea of it except the mechanical side" (*Dental Cosmos*, November, 1922, p. 1208).

Dr. Murphy, addressing the Chicago Dental Society in 1916, asks: "Why should the dentist be educated in a different room from the surgeon in the essential elements—in his bacteriology, histology, biology, physiology and anatomy? He should not. It is an anomaly. The first two years, as indicated by Dr. Thornton, should be the same with the aurist, the oculist, the surgeon, the neurologist, the internal medical man and the dentist, and until this educational error is corrected there will be no correlation and no coöperation between dentistry and internal medicine. . . . Another proposition, it appears to me, is that after we have educated the dentist and physician together; after we have practised side by side, each one will see, as he gleans knowledge from the other, a new method of advancement" (Proceedings of the Chicago Dental Society, Fifty-second annual meeting: *Dental Review*, January 28, 1916, p. 381).

The latest word which has come to my attention is from Dr. W. S. Thayer of Johns Hopkins University, who wrote recently to the dean of the medical school at Minnesota: "The close association of dentistry with medicine and surgery is a matter which I have very much at heart. I think it is most unfortunate that the instruction of the dentist is in any way different from that of the doctor and surgeon. Dentistry should be just as much a specialty of surgery and medicine as ophthalmology, otology, rhinology or any of the other well-recognized specialties." Columbia, as you know, approaches closely this ideal of a joint training, with its requirement of two years of pre-dental work. The University of Rochester is about to inaugurate a school of dentistry with a requirement of three pre-dental years of college work.

With the long desired survey of dental education an accomplished fact, future progress in the right direction is made certain. When we consider that we are emerging from the long upward struggle onto a pleasant field of assured usefulness, we think back to the great spirits of all ages who have handed on the torch, and realize with Goethe that

> "All that has worth lives in the Past,
> Enduring forever in splendid activity."

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